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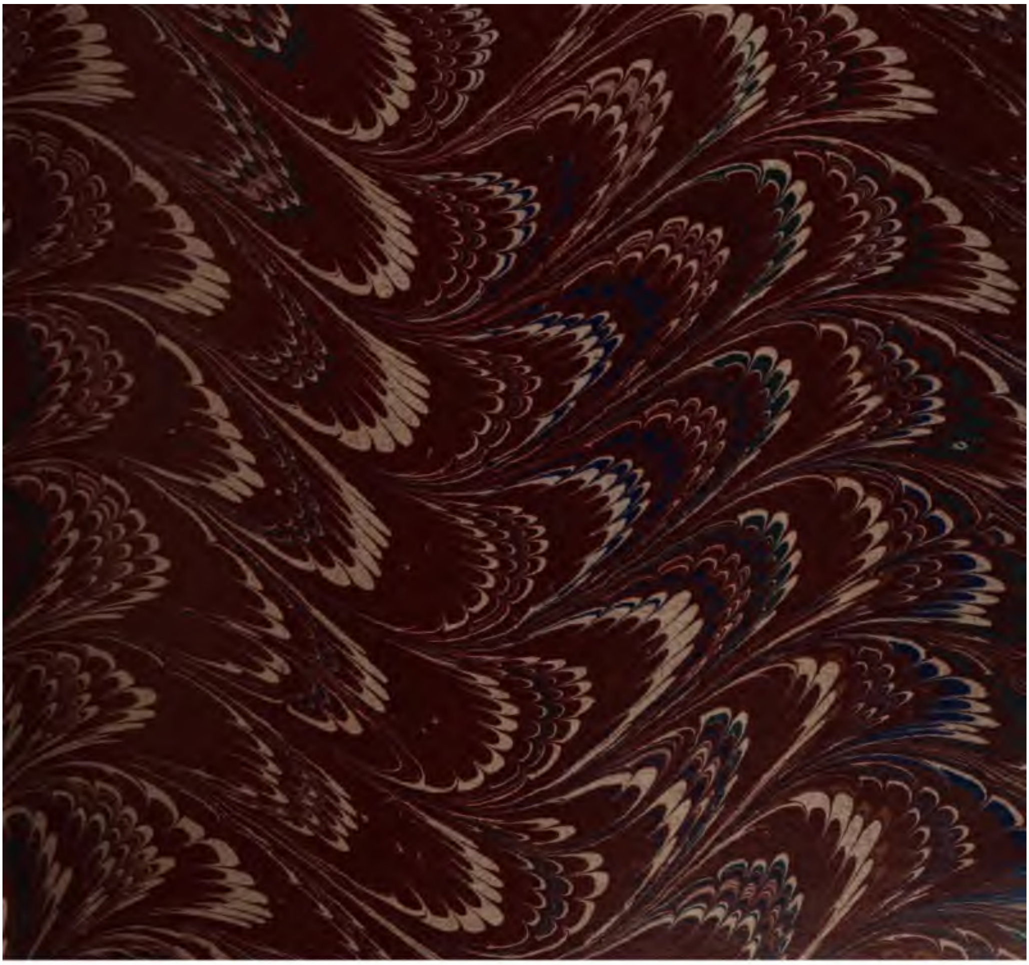

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PRACTICE OF MEDICINE.

EDITED BY DR. H. VON ZIEMSSEN,
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VOL. XIII.

LIBRARY

DISEASES OF THE SPINAL CORD
AND
MEDULLA OBLONGATA.

BY

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(Translated by Edward G. Geoghegan, M.D.)

DISEASES

OF THE

SPINAL CORD AND ITS ENVELOPES.

ERB.

INTRODUCTION.

OUR knowledge of the diseases of the spinal cord is rapidly extending. This numerous, important, and interesting class of maladies, long neglected, is at present receiving the profound attention of various classes of observers, and the labors bestowed upon it are bearing fruit in many ways.

This improvement forms but a part of the general forward movement of the last twenty or thirty years, in which special pathology has shared equally with other branches of scientific medicine. There are, however, three sources from which our knowledge of the diseases of the spinal cord has been especially enriched.

First in order, and most important, stands the improvement in the *experimental physiology of the spinal cord,* which has been made within the period of twenty or thirty years. The study of this subject has led to extremely remarkable and important results, many of which yet remain a matter for controversy or doubt. Unexpected, even insurmountable difficulties, have risen to obstruct what we at first thought an easy quest; but these difficulties have served only as incentives to deeper and fuller investigations, which have proved abundantly fruitful of isolated facts, often of the greatest value in pathology.

The *improved methods of pathologico-anatomical examination* have been equally important in developing our knowledge of the diseases of the cord. Although in use for little more than ten years, they have been constantly receiving improvements, and have already added greatly to our knowledge and comprehension of the facts. By their means many diseases have been discovered, whose existence was not even suspected by those using the former imperfect methods of examination; through them a hitherto unknown degree of exactness in localizing disease has been rendered possible, and we have learnt to refer numerous morbid phenomena to definite local changes in the cord.

Conjoined with physiological research and an improved, intelligently directed method of clinical examination, the study of pathological histology has thrown great light upon the general pathology of the spinal cord, and has led to the most interesting conclusions in respect to pathological as well as physiological processes.

Finally, we may mention the *progress of therapeutics*, which has lent the impulse of a new interest to the study of many forms of disease of the spinal cord. We have lately learnt to cure a number of such diseases, formerly thought incurable; or, at least, we have been enabled to improve the prognosis in many such. This we mainly owe to the science of electro-therapeutics, which has made so many contributions to the pathology of the nervous system; not a few of the advances that have been made in spinal pathology are associated with the names of electro-therapeutists. Nor is balneo-therapeutics (the scientific aspect of which has greatly improved of late) less deserving of credit in this respect.

The diseases of the spinal cord have thus come to furnish a most attractive and interesting field for scientific research, and one most fruitful of important practical results; we can say that the progress made in its cultivation has been of late years extremely satisfactory.

And yet it must be stated with emphasis, that we only stand at the beginning of a successful development of the subject, and that an extremely large amount of work remains to be done.

We cannot help seeing that the rich results of physiological research are still very defective and uncertain in many essential points; the results, indeed, often change from day to day with each new method and new observer, and they are far from always possessing that degree of exactness and trustworthiness to which physiological research would lay claim. The excessive difficulty of the subject explains why it is that in many points we have perhaps not yet reached the root of the matter.

Nor is it less certain that the researches and results of pathological anatomy are still far from trustworthy, that the points thoroughly explained are but few, and that the general pathological significance of the commonest and most important morbid processes in the spinal cord is not yet clear. The impossibility of observing with exactness all the characters of a fresh spinal cord, the errors and uncertainties attending the examination of hardened specimens, and finally, the undeniable fact that these researches have given us no information at all in regard to not a few diseases, or stages of diseases, of the cord—these circumstances ought to make us very cautious about viewing these diseases as if their key were held by pathological anatomy alone.

Practical experience, finally, shows us that the treatment of diseases of the cord is still without hope in many respects. The number of desperate cases which mock at all kinds of treatment shows us, with continually fresh emphasis, how much remains to be discovered and achieved.

All the more encouraging is the way in which the work is going forward. Numerous investigators are busy with the physiology and pathology of the spinal cord; every day brings fresh discoveries, new additions to our knowledge, a broadening and clearing of our views.

It is obvious that it is extremely hard to write a text-book of diseases of the spinal cord in the midst of this crowding rapidity of growth of knowledge. It is perhaps impossible to state in any decided terms the daily changing position of our knowledge; and a dogmatic statement like the present, compelled as it must be to shun the prolixity of a monograph, and to refrain from the thorough discussion of debatable questions, requires every species of caution in its presentation.

The preceding remarks will make it sufficiently plain why we consider the clinical point of view as the most important one at the present time. We write for the practising physician, who encounters the actual diseases from day to day. For him it is important that a description should possess unity and clearness, and should give him something to work by. We have therefore laid the chief stress upon the clinical presentation, in making which we have rested mainly upon the basis of pathological physiology, bestowing meanwhile a due share of attention upon pathological anatomy.

One thing more should be added. After long deliberation, we have decided that the special descriptions of disease should be preceded by a general account, which has grown to a great size, but will not, we hope, be found superfluous or worthless.

The anatomical introduction, the brief statement of the macroscopic and microscopic anatomy of the cord and its membranes, with a synopsis of the physiology of the cord, which we have placed first, are justified by the facts that these things are indispensable to the understanding of the diseases; that they mostly escape the memory of the practising physician in the course of time; that they have to be sought in text-books and journals which are not often possessed by practising physicians, and that, even in such works, they are not always treated with regard to the pathology, nor even with a proper understanding of the latter.

An account of the general symptoms seemed to us most desirable in aiding the student to understand the pathological phenomena, and in saving repetitions and explanations in the special division of the work. We have, therefore, taken pains to present in this part a brief and clear account of researches in physiology and pathological anatomy, and of clinical observations, pointing out by the way the chasms which exist in our knowledge.

Finally, it seemed not undesirable to describe the general treatment, especially as regards electro-therapeutics and balneotherapeutics—very important branches, which, at the present time, have hardly received a connected scientific treatment; although, even here, we have had to point out many defects and obscurities—perhaps more than had been anticipated.

I. Anatomical Introduction.

Cf. *Longet*, Anat. et physiol. du système nerveux. German transl. by Hein. 1847.—*Koelliker*, Mikroskop. Anatomie. Handb. der Gewebelehre, 5. Aufl.—*Stilling*, Neue Untersuchungen über den Bau des Rückenmarks. 1857.—*Bidder* und *Kupffer*, Untersuch. über die Textur des Rückenmarks u. s. w. 1857.—*Schroeder van d. Kolk*, Bau und Function der Medulla spinalis und oblongata. Braunschweig, 1859.—*Goll*, Denkschrift der med.-chir. Gesellsch. des Cantons Zürich. 1860.—*Frommann*, Untersuch. über die normale u. pathol. Anatomie des Rückenmarks. 1864.—*Deiters*, Untersuch. über Gehirn und Rückenmark des Menschen u. s. w. 1865.—*M. Schultze* in Stricker's Handb. der Gewebelehre. —*Gerlach*, ibidem.—*Henle*, Handb. der Anatomie. III. Bd. 2. Hälfte.—*Wundt*, Physiologische Psychologie. Leipzig, 1874.—*C. Lange*, Ueber chron. Rückenmarksentzündung. Kopenhagen, 1874; see Schmidt's Jahrb. Bd. 168. p. 238. 1875.—*Leyden*, Klinik der Rückenmarkskrankheiten. I. 1874.—*Huguenin*, Allg. Pathol. der Krankheiten des Nervensystems. 1873.—*Boll*, Histiologie und Histiogenese der nerv. Centralorgane. Arch. f. Psych. und Nervenkrankheiten. IV. p. 1. 1874.—*Schiefferdecker*, Beitr. zur Kenntniss des Faserverlaufs im Rückenmark. Arch. f. Mikroskop. Anatomie. X. 1874; and numerous other authorities.

The *spinal cord* is suspended in the vertebral canal, hanging almost free, and with a considerable degree of mobility.

It is unnecessary to describe the vertebral canal in this place. As points of practical importance, it may be stated that its anterior wall is perfectly firm and solid, being composed of the bodies of the vertebræ, with the interposed disks of cartilage, while its posterior and lateral walls have numerous vacant spaces, which are only filled up by ligaments and other soft parts, as nerves, blood-vessels, etc. The lateral spaces (intervertebral foramina) exist along the entire canal; the posterior, on the contrary (intervertebral fissures), are distinctly marked in no place, except in the cervical region—particularly at the two uppermost vertebræ—and again from the tenth dorsal vertebra downward, especially in the lumbar part. Throughout the greatest part of the dorsal vertebral column these fissures are completely closed by the vertebral arches, which cover each other like the tiles of a roof. From this it is manifest what portions of the cord[1] are most exposed to external injuries.

The cord and its envelopes are far from filling up the entire canal; and this circumstance protects the cord from injurious pressure in the most movable parts of the column, namely, the cervical and lumbar regions. The width of the canal varies; it is greatest in the regions of the neck and loins, least in the dorsal region, especially from the sixth to the ninth dorsal vertebra; within the sacrum it rapidly diminishes. The shape of its transverse section is nearly circular in the dorsal region, while in the cervical and lumbar regions it is drawn out laterally, and assumes nearly the form of an obtuse-angled triangle, with its basis directed forward; in the sacrum it has the form of a half-moon, with the convexity directed backward.

The length of the cord is much less than that of the canal. Its extreme tip (the end of the conus terminalis) lies in adults at or near the boundary between the first and second lumbar vertebræ. Fehst[2] asserts that a difference exists between the sexes in this respect; in men, the lower edge of the first, in women, the lower edge of the second lumbar vertebra forming the extreme limit of the cord.

It is a matter of some practical importance to be able easily to distinguish the different regions of the canal, in order to define with accuracy the location of a given disease; this is done by palpation and counting the spinous processes. Thus, we easily recognize the spinous process of the second cervical vertebra, and that of the seventh (vertebra prominens), from which points the separate processes can easily be counted by palpation. It is not so easy to recognize the spinous process of the twelfth dorsal by the insertion of the twelfth rib.

The greatest part of the cavity of the canal is lined with a hard periosteum, covering the bony walls in every part.

Within the canal, the cord is covered, first, by a cylindrical fibrous sac of wide dimensions, the dura mater spinalis, which begins at the foramen occipitale magnum, to the edges of which it is firmly attached, and terminates by contracting itself around the filum terminale, finally losing itself in the periosteum of the coccyx. The outer surface of the dura is not closely connected with the walls of the canal, but is separated from them by a loose, moist, connective tissue, with abundance of fat, which envelops every part of the dura in a layer of greater or less thickness. The inner surface of the dura is smooth and shining, and covered with several layers of pavement epithelium. The neurilemma of the nerve-roots which perforate the sac of the dura unites with the tissue of the latter.

---

[1] Spinal cord: the term will be thus used henceforward.

[2] Centralbl. f. d. med. Wissensch. 1874. No. 47.

The dura is supplied with arterial blood by the vertebral, intercostal, and lumbar arteries; its venous blood passes off by veins which form large plexuses in the loose cellular tissue of the anterior and posterior surfaces of the dura, connected with the external vertebral plexuses. Numerous nerve-fibres are supplied to the tissue of the dura and the periosteum of the canal.

The *pia mater spinalis*, the so-called vascular coat, lies much closer to the cord. It envelops the cord in the closest manner from top to bottom; it forms an exactly fitting cylindrical sheath for the cord, containing the blood-vessels that belong to it, and closely united in every part with it; it sends numerous sheath-like processes into the interior of the cord, which divide into many branches, spreading in all directions among the nervous elements of the cord, forming a framework for the support of these elements, and imparting to the cord a due degree of firmness; the largest of these processes, easily visible to the naked eye, lies in the anterior median fissure of the cord, while a smaller one is seen in the posterior fissure; but numberless finer processes pass into the substance of the cord from the entire periphery of the pia.

The pia mater is a membrane of connective tissue of considerable toughness and firmness; it consists almost wholly of waved connective tissue, is extremely rich in blood-vessels (which will be further described in speaking of the blood-supply of the cord), and is also rich in nerves, which originate from the posterior roots. Often, especially in old people, the pia is strikingly rich in pigment, so as to have a light gray or brownish tint; this is commonest at the cervical part, and is by no means always pathological.

The pia is joined to the dura on either side by from twenty to twenty-three processes of a three-cornered shape; they are arranged in two perpendicular rows, one on each side of the cord, with their bases inserted into the pia, and their points into the dura. They constitute the ligamentum denticulatum.

The pia accompanies the filum terminale to the end of the canal, and is there united with the dura and the periosteum of the coccyx.

Between the dura and the pia lies the *arachnoid*. Henle describes it as an unusually loose, watery, areolar tissue, which becomes compacted in the direction of the dura to a connected, delicate, resistant layer (*arachnoid proper*), while its inner portion passes directly into the tissue of the pia. Between the inner and the outer thickened layers (viz., the pia and the arachnoid) there exists, therefore, a loose areolar tissue, which is designated, and with propriety, as *subarachnoid tissue.*

The fluid which this tissue contains in abundance is of great importance, and forms that part of the *cerebro-spinal* fluid which is contained in the spinal canal. It is clear, contains but a small amount of solids, and appears poor in microscopic elements. Its quantity in the adult amounts to about sixty grammes, but varies considerably. It exists under a certain positive though moderate pressure. When the dura is pierced and the arachnoid also injured, it flows off.

The function of this fluid is, without doubt, that of protecting the cord from mechanical injuries; by keeping it floating in a fluid, it preserves as equal a pressure as possible, and perhaps also regulates the circulation and the pressure in the blood-vessels. A sudden evacuation of this fluid in cases of injury to the dura is followed by serious disturbances, which are, however, doubtless due in part to cerebral implication.

The spinal fluid is not in a state of repose, but, as Quincke[1] has recently given exact proof, is constantly undergoing a double motion: first, it moves to and fro in the subarachnoidal tissue, under the influence of respiration; and second, it is continually secreted under a definite pressure by the blood-vessels, and as continually passes off by certain channels into the lymphatic vessels. These channels for the spinal fluid are chiefly situated in the nerve-trunks which leave the spinal canal. It is obvious that these motions in the spinal fluid may be of the greatest importance in the propagation of morbid meningeal processes, in the removal and transportation of inflammatory and other products.

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[1] Zur Physiologie der Cerebrospinalflüssigkeit. Reichert's und Du Bois-Reymond's Archiv. 1872. Heft 2.

The *spinal cord* (medulla spinalis) is a cylindrical cord, somewhat flattened in front for a great part of its length, and not in every part of equal thickness. It does not nearly fill the sac of the dura, but is closely wrapped by the pia. After removal of the attached roots, it is easy to discern two swellings, the *cervical enlargement* and the *lumbar enlargement.* While the thinnest part of the cord, that in the dorsal region, has a transverse diameter of about 10 mm., and a sagittal diameter of about 8 mm., the cervical enlargement measures 13 or 14 by 10 mm., and the lumbar enlargement 12 by 9 mm. The diameter of the upper cervical region remains about 11 or 12 mm.

The spinal cord begins where the medulla oblongata terminates, but without any sharp line of demarcation. Its uppermost limit is best placed just above the point of exit of the first pair of cervical nerves, lying at about the height of the upper edge of the posterior arch of the atlas. Its conical tip (conus terminalis) is situated opposite the body of the first or second lumbar vertebra. The lumbar enlargement reaches from the beginning of the conus terminalis upward to the tenth dorsal vertebra; the cervical enlargement, from the second dorsal upward to near the middle of the cervical column, at the third or fourth vertebra. The lower end of the conus terminalis is prolonged into the filum terminale, which reaches to the end of the canal. The average length of the cord is from 35 to 40 cm.

The consistency of the cord varies somewhat in individual cases; in the perfectly fresh state it is quite tough and elastic, and easy to cut; its cut surface is then smooth, and seldom pushes over the edges; shortly after death it begins to grow soft and deliquescent, and thus offers much greater obstacles to examination.

Besides these features, we observe in the cord a number of grooves, which, even when seen from without, give a hint of the internal construction of the cord.

On the somewhat flattened anterior aspect, we find the anterior median groove running down the whole length of the cord, which, sinking into the cord, forms the broad fissura longitudinalis anterior,[1] reaching nearly to the centre of the cord, and containing a large process of the pia mater.

On the posterior aspect a similar groove runs from top to bottom, the posterior median groove, which, in like manner, sinks to a fissura longitudinalis posterior,[2] directed in the sagittal line towards the centre of the spinal cord. This fissure also contains a process of the pia mater, though a much smaller one, closely attached to the adjacent walls of the fissure.

These two fissures divide the cord in two symmetrical lateral halves, which are united by a narrow bridge composed of the white and gray commissures. The anterior fissure is broader than the posterior, but shallower.

After these fissures, the first thing to observe are the nerve-roots which leave the cord, arranged in a double row, one on each side. The posterior root-fibres lie in a perpendicular line above one another, and form an almost continuous row, which maintains a given distance from the posterior median fissure, but gradually approaches it as they descend. If all the root-fibres are removed, their points of exit form a sort of longitudinal groove, called the *sulcus lateralis posterior*, or posterior lateral fissure.

The anterior root-fibres do not leave the cord in a single row, but are scattered over a stripe about 2 mm. broad, on the anterior portion of each side of the cord. Their distance from the anterior median groove is also definite, but by degrees becomes less. When the root-fibres are removed, this stripe is plainly marked, and is designated as the anterior lateral fissure—*sulcus lateralis anterior*.

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[1] Anterior median fissure of Quain.

[2] Posterior median fissure of Quain.

In the upper half of the cord another fissure or groove is visible, lying about half-way between the posterior median and the posterior lateral fissures, and designated as the *sulcus intermedius posterior*.

These fissures are commonly used to bound the regions of the white substance of the cord. In each lateral half, the white mass lying between the anterior median and the anterior lateral fissure is called the *anterior column;* the mass lying between the anterior and the posterior lateral fissure is called the *lateral column;* and the white matter between the posterior lateral and the posterior median fissure is named the *posterior column.* In the upper division of the cord, this posterior column is again divided by the sulcus intermedius posterior into two columns, which are genetically quite distinct (Pierret), and which claim a very special significance in pathology; the median portion of the posterior column, adjacent to the posterior median fissure, is called the *wedge-shaped column of Goll*, or *funiculus gracilis* (zarter Strang); while the lateral portion, which adjoins the posterior lateral fissure, is called *funiculus cuneatus* (Keilstrang).

Most of these divisions of the white mass of medulla are more or less arbitrary; the finer anatomy of the cord recognizes only the sharp division of the anterior and posterior median fissures. Pathological facts, however, not only justify the above division of the posterior cords, but permit a division, not sharply made, into *outer and inner anterior column*, and *posterior and anterior lateral column.*

The *anterior and posterior nerve-roots* arising from each lateral half of the cord converge, and, after uniting, pierce the dura and pass to the intervertebral foramina; the posterior root of each spinal nerve enlarges to a ganglion (*ganglion spinale*) before entering the foramen, while the anterior root passes by this ganglion and unites farther on with the posterior root to form a spinal nerve. The distances between the origins of roots being less than those between the intervertebral foramina, the lower roots are compelled to take a more and more oblique course in order to reach the foramina; those from the conus terminalis run almost parallel, forming a bundle of nerves descending within the sac of the dura mater, named *cauda equina.*

This is nearly all that can be seen upon external inspection of the cord.

In studying the inner structure of the cord it is necessary, above all, to attend to cross-sections; they give the best information, and by mentally joining the results of the various sections for the whole length of the cord, a correct idea is gained of the peculiarly complicated columnar structure of the cord.

In any section we may make we recognize first a division into two substances: the central gray or gray-reddish mass, irregularly defined, and of a peculiar shape, generally resembling that of the letter H, called the *gray substance;* and a peripheral white mass surrounding the gray, filling in the irregular outlines of the latter, and giving to the whole the form of a cylinder, called the *white substance.* The latter is divided into the above-named columns by the fissures and the nerve-roots, a division which, in the lateral parts, is distinct only upon the surface, and does not extend clearly into the mass of the white substance.

When we look closely at a section of the cord, we discover in its centre a fine canal, often filled with tissue elements or pathological products—the *canalis centralis.* This canal opens upward into the fourth ventricle; at the lower end of the cord, at the tip of the conus terminalis, it expands to a small cavity—the ventriculus terminalis lately described by Krause[1]—which again opens into the cavity of the filum terminale.

The central canal is surrounded by a partly gray and partly white mass, which joins the two halves of the medulla; the gray portion of this bridge, in which the central canal lies, is behind, and is called the *gray* or *posterior commissure;* the white portion lies in front, borders directly upon the anterior median fissure, and is called the *white* or *anterior commissure.* See Fig. 2, *r* and *q*.

From this median region the gray matter extends in considerable mass and peculiar form into each half of the medulla. Its anterior part is rounded and broad, and pushes towards the antero-lateral column; its posterior part is narrower and more pointed, and points directly to the posterior lateral fissure; it bounds the posterior columns externally, and separates them from the lateral columns. The two parts of the gray substance are connected; they are bounded outwardly by a line, which is, in general, concave on its outer side. But this line is irregularly pressed outward in many places by projecting masses of gray matter, and has scarcely the same form in any two sections. See Fig. 1.

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[1] Centralbl. f. d. med. Wiss. 1874. No. 48.

The anterior rounded part of the gray substance is called the *anterior cornu*, or, better, when considered as extending through the entire length of the cord, the *anterior pillar;* the posterior acute portion is called the *posterior cornu*, or, better, the *posterior pillar*. Subsequent examination will show that the gray substance differs very much in elementary composition at different heights.

This may be seen, without the aid of a microscope, by observing that the form of the transverse section of the gray matter differs extremely in different sections of the cord, as a glance at Fig. 1 will show.

The section made in the dorsal region is the smallest, and most resembles a Roman H, with its fore limbs rounded and its hinder ones pointed (Fig. 1; 4 and 5); in the cervical and lumbar enlargements it is much thicker, is provided with rounded horns, swelling into heads, and is enlarged by deposits of various shapes (Fig. 1; 2, 3, 6, 7). There is no doubt that these enlargements of the spinal cord are chiefly, if not exclusively, due to the increase in the gray substance.

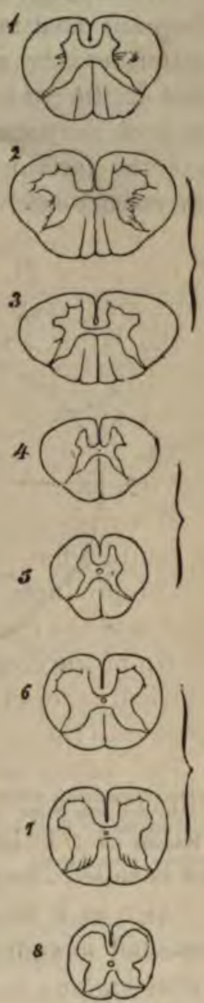

FIG. 1.

Sections of the human spinal cord taken at different heights. Magnified $^3/_2$.
1. Upper cervical portion.
2,3. Cervical enlargement.
4, 5. Dorsal portion.
6,7. Lumbar enlargement.
8. Conus terminalis.

In many portions of the cord various processes are seen to leave the lateral portions of the gray matter, usually containing bundles of nerve-fibres, which radiate to various depths in the white matter; these processes meet and form a kind of network, which encloses separate portions of the white columns, rendering the outer boundary of the gray matter very uneven and jagged.

These gray bundles of fibres are called *processus reticulares*. They are most marked at the boundary between the anterior and posterior cornua (Fig. 2, *p*). In the cervical and upper dorsal part of the cord a triangular prismatic process projects from the basis of the anterior cornu in front of these reticular processes into the lateral column, which has been designated the *tractus intermedio-lateralis* (Fig. 2, *o*).

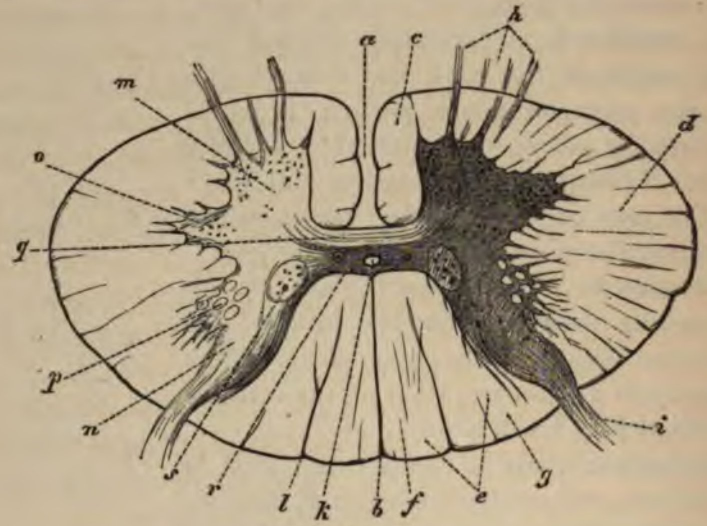

FIG. 2.

Semidiagrammatic section of the cord about the lower part of the cervical enlargement. Magnified $^{8}/_{1}$. *a*. Anterior median fissure. *b*. Posterior median fissure. *c*. Anterior column. *d*. Lateral column. *e*. Posterior column. *f*. Goll's wedge-shaped column (Zarter Strang). *g*. Funiculus cuneatus. *h*. Anterior roots. *i*. Posterior roots. *k*. Central canal. *l*. Sulcus intermedius posterior. *m*. Anterior pillar (cornu). *n*. Posterior pillar (cornu). *o*. Tractus intermedio-lateralis. *p*. Processus reticulares. *q*. Anterior or white commissure. *r*. Posterior or gray commissure. *s*. Clarke's column or columna vesicularis.

An exact idea of these forms is only to be gained by the repeated examination of good sections from the hardened cord, under various powers of the microscope.

All the unevenness, indentations, faults in the contour of the gray matter, are filled out by the *white substance*, which surrounds the gray in a layer of various thickness like a cloak, and gives to the cord its external form of a more or less perfect cylinder. The mass of white substance diminishes slowly but perceptibly in the downward direction (Gerlach); it disappears wholly at the commencement of the filum terminale.

The white substance is traversed by numerous radiating *septa* of various degrees of fineness, and by the nerve-roots, which cross it from the gray matter to the surface in a similar radiating manner. The septa are connected by numerous branches, which divide up the field of white substance into numerous rhombic districts of various size, in which the nerve-fibres of the white substance are contained. The septa and septula thus compose a fine and complex network, enclosing the vessels of the cord and the nerve-fibres.

At its outer border, just under the pia mater, the white substance is again enclosed by a very fine layer of gray. This sursounds the white substance like a thin overcoat, accompanies the processes of the pia which enter the cord, and separates the nerve-bundles proper from the septa; it sends out from the septa numerous offshoots, which penetrate among the nerve-fibres and envelop each one completely. Most later observers are agreed in considering that this substance is almost, if not entirely, composed of the connective substance of the cord (neuroglia).

Comparing now the sections with each other, we obtain the following *plastic view of the structure of the cord:*

Its nucleus is formed by a pillar of gray matter which traverses the whole length of the cord. This may be approximately compared with a channelled column of somewhat irregular form, perforated by a fine canal in its centre, and possessing four chief projections and four intervening depressions. This pillar is thin and slender everywhere, but is made thicker by accessions of new matter in the cervical and lumbar regions.

Its forward projections are rounder, broader, more massy, forming the anterior cornua; its posterior projections are sharper, more slender, narrower, forming the posterior cornua. The nerve-roots pass off from each of the four projections in the form of fringes.

Of the four depressions, the anterior and posterior are the deeper, smoother, and more regular; the two lateral are shallower and less regular; their bases are made irregular everywhere by the deposition of gray masses, by projections and knobs upon the pillar; in parts they are wholly filled up, and here and there a longitudinal ridge comes out into them.

The white substance is as it were pressed into these channels, as when a soft clay is used to fill up the flutings of a pillar and smooth over its irregularities. Being smoothed off exteriorly, these white masses finish the round outline of the column. The white substance may also be conceived of as composed of long strings or ribbons which fit exactly into the existing cavities and spaces, and are laid into the channels of the central gray pillar.

The whole is then covered in with a fine gray mantle, which closely embraces the cord, as does the pia mater.

Blood-vessels and lymphatics of the cord. The tissue of the cord is rich in blood-vessels; the gray substance possesses an especially rich capillary network. All the blood-vessels arise from the pia; they pass into the cord through the processes of the pia, and, following their ramifications, send an abundant network of capillaries into the gray and white substance.

The arteries of the pia originate in the vertebral arteries. Each vertebral artery gives off an anterior and a posterior spinal artery. The two anterior spinal arteries unite in a single stem, which runs down along the whole length of the cord without much loss of diameter to the conus terminalis; this arteria impar is reinforced at the level of each pair of nerve-roots by small arteries which originate in the intercostal and lumbar arteries, and pass through the intervertebral foramina with the nerve-roots; on the other side it distributes numerous fine twigs to the pia and cord, and finally, at the foot, it forms two anastomoses with the posterior spinal arteries.—The posterior spinal arteries, one on each side, run down underneath the posterior roots, with each pair of nerves receiving fine branches of communication from the intercostals, and sending off numerous fine twigs to the pia and the cord.

It therefore seems plain that the pia mater and cord are principally nourished by the vertebral arteries, and next to them chiefly by the intercostals.

The capillaries of the cord discharge their blood directly into two central venous trunks, lying to right and left of the central canal within the gray commissure (Fig. 2), and running the whole length of the cord. By their numerous horizontal connections they distribute their blood to the outer veins of the cord; of these the greatest and most important is the vena mediana spinalis anterior, which runs the whole length of the cord, lying behind the anterior spinal artery in the anterior median fissure. Down the posterior median fissure runs the vena mediana spinalis posterior, which increases gradually in size as it descends. Numerous venous networks, which also increase in calibre as they descend, unite these external veins to each other. The latter transmit their blood (through branches which run with the nerve-roots and pierce the dura) to the great spinal plexuses, which lie in the loose fatty tissue surrounding the dura, and anastomose with the outer vertebral plexuses, etc.

Regarding the *lymphatic passages* in the cord, little is known exactly. The perivascular lymph-spaces first carefully described by His (and since more fully by Boll and Adler—see Archiv f. Psychiatrie u. Nervenkr. Bd. IV. and V.), are said also to exist in the cord. They stand in connection with a large lymph-space, lying between the pia and the cord, from which the lymph is carried through the lymphatics of the pia. According to Schwalbe the subarachnoidal space is also a lymph-space, but not directly connected with the perivascular lymph-spaces of the cord.

Finer structure of the cord. The cord is composed of very various tissue-elements, for the exact histological description of which we refer to hand-books of histology; as regards the finer structure of the important nervous elements, compare the article by M. Schultze in "Stricker's Manual of Histology." In this place only a brief sketch can be given.

Nerve-fibres, both medullated and non-medullated, occur in the spinal cord. All these are completely destitute of Schwann's sheath, or, at all events, it has not been demonstrated by the present methods of research. The medullated fibres compose the greatest part of the white substance and the anterior commissure; they occur of very various diameter, the thickest being found in the anterior columns; the funiculi graciles contain only slender fibres. In all, the axis cylinder may plainly be seen in cross-section, and its diameter is nearly proportioned to that of the medullary sheath. The finest medullated fibres are found in the gray matter, of which they form a preponderant element. They pass in all directions through the gray matter, both isolated and in bundles, and frequently dividing up. The non-medullated fibres, analogues of the naked axis cylinders, are only found in the gray matter, in which they ramify to a very great extent; their finest branches unite to form a close mesh-work, which, with the ganglion-cells, is especially characteristic of the gray substance (Gerlach). And a few bundles of larger fibres with medullary sheaths, originating in the nerve-roots, pass through the gray substance for short distances.

The cellular nervous elements, the *ganglion-cells*, are found almost exclusively in the gray substance, and are the component which most strikes the eye; when they are found in the white substance, they are almost always single, and close to the gray.

They are large, multipolar cell-bodies, sometimes visible to the naked eye; they have no envelope, their nucleus is large, with a distinct glistening nucleolus, and they usually contain an accumulation of pigment granules. They are remarkable for their numerous radiating processes, almost all of which ramify abundantly (protoplasm processes), while one process (the nerve-process) remains smooth and undivided, and after a longer or shorter course receives a medullary sheath and becomes a medullated nerve-fibre. This undivided process is, therefore, also called the axis cylinder process.

According to Gerlach, not every ganglion-cell possesses a nerve-process, but many are connected with the fine network of nerve-fibres only through the ramifications of their protoplasm processes; among such, the smaller forms of cells occurring in the posterior cornua are especially named. These cells, therefore, are connected with nerve-fibres only through the intervention of the fine network of nerves. Boll, however, does not consider that the existence of this kind of cells is sufficiently established.

The size of the ganglion-cells in the cord, like their form, is very variable. They are found small, medium-sized, and large. By far the largest are found in the anterior cornua, with a great number of processes; the smallest, more of a spindle shape, in the posterior cornua; those of medium size and a more rounded form, in the so-called columns of Clarke.

They lie in groups and heaps, forming, in various parts of the gray substance, actual columns of cells extending for a considerable distance in the cord; they are especially abundant and definite in their distribution in the gray anterior cornua, while in the posterior cornua they occur but sparingly and in quite irregular arrangements.

The attempt has often been made to establish a close relation between the form and size of the ganglion-cells and their functions. Jakubowitch first stated expressly that the larger cells with many processes, in the anterior cornua, are motor cells, while the smaller should be regarded as sensitive, and the smallest, of a spindle shape, as sympathetic (vaso-motor). Other observers have made similar hypotheses, and, more especially of late, a trophic action has been ascribed to the ganglion-cells, upon the basis of pathological facts. Of all this only so much appears certain: that the large cells with many processes in the anterior cornua are most intimately related to the motor apparatus; but what these relations are, and how they are expressed in the form and size of the cells, in their position and grouping, is quite unknown at present; nor is anything whatever known with certainty regarding the existence, position, form and size of "sensitive," "vaso-motor," "trophic," "reflectory," "automatic" ganglion-cells, although many pathological facts recently collected seem to give us at least a starting-point for the solution of these problems.

The nervous fibres and cells, which are certainly the most important components of the cord, are enveloped in a basement substance of connective tissue, the so-called neuroglia, which gives support and firmness to the cord. This substance radiates inward from the pia in numerous septa, bearing the blood-vessels, which break up into a great number of branches, and finally form a very delicate network in which the nervous elements are embedded. The gray cortical layer and the greatest part of the trabecular network proceeding from it, a great part of the gray substance (especially that which is commonly named the substantia gelatinosa), and the entire supporting framework of the white substance, are composed of this neuroglia. In regard to its finer structure, the opinions of the best authorities (Koelliker, Frommann, Gerlach, Henle, Boll, Ranvier, C. Lange, and others) are still divided, as the difficulty of investigating this tissue is very great. All observers agree that the neuroglia consists chiefly of a network of fibres very closely interlaced, embedded in a more or less abundant finely granular basement substance, and containing numerous nuclei, granules and cell-elements. But, as regards the significance and the exact character of these fibres and cells, a great deal of controversy still exists. Some consider the fibres as elastic (Gerlach), others as more like connective tissue (Henle, Ranvier), others as the processes of cells (Kölliker, Boll, C. Lange); and the interpretations of the interjacent cellular and nucleoid structures are equally various.

Boll has very recently given a careful description of the neuroglia, which is different from previous ones. He states that its sole component is a form of multipolar connective tissue cell, composed of countless fine processes, not ramified, and a nucleus. These cells envelop the vessels in the septula, like sheaths, then branch off from the sheaths, and at last form by themselves the septula which embrace the single nerve-fibres and groups of fibres—as finely woven twigs embrace the upright staves of a basket, or as the fingers of the two hands may be woven into a kind of tube. These peculiarly shaped cells, characterized especially by their numerous unbranched processes, are called Deiters' cells or the spider-cells of Jastrowitz, and are often particularly easy to see in pathological specimens of the cord. Among their processes there lies a small quantity of a granular interfibrillary substance. Their nuclei, according to Boll, are what Henle calls "granules" (Körner). An essentially similar description of the neuroglia is given by C. Lange.[1]

Very recently, Ranvier has presented another view, according to which the connective tissue of the spinal cord is quite analogous to the interstitial connective tissue of the peripheral nerves. It is said to consist of numerous fine fibrillary bundles of connective tissue, not anastomosing, but crossing in many directions, and at the points of crossing there lie flat nucleated connective tissue cells.[2] It is not necessary here to decide these difficult histological points; it is enough to know that the entire spinal cord, both white and gray matter, is permeated by a fine-meshed structure of connective tissue, consisting chiefly of fine fibrils in which numerous nuclei are embedded, and surrounding in the closest manner the nervous fibres and cells.

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[1] See Virchow-Hirsch's Jahresbericht for 1873, Bd. II. p. 76, and Schmidt's Jahrb. Bd. 168. p. 239. 1875.

[2] Centralblatt f. d. med. Wiss. 1874. No. 31.

If Boll's view should turn out to be the true one, the "granules" of Henle would at last receive their right interpretation. They have been looked upon in all possible lights: as young connective-tissue cells, as young nerve-cells, as lymph-cells, as wandering colorless blood-cells without prospect of future advancement, etc. But it cannot be denied that not a few structures resembling cells and nuclei are found in the cord, whose exact interpretation is not easily made out.

The arrangement of the histological elements of the spinal cord is extremely complicated, and it is very hard to make it out accurately in all its details and in all parts of the cord.

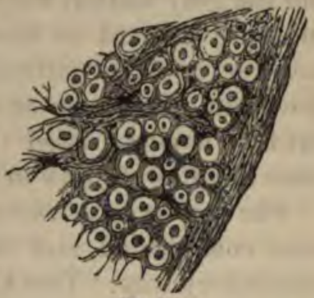

FIG. 3.

A piece of the transverse section of the white substance of a lateral column. Neuroglia, with Deiters' cells embedded in it, embraces the nerve-fibres, which are cut across, and are seen with distinct axis-cylinders. Magnified $\frac{220}{1}$.

Very simple by comparison is the *structure of the white columns*. They contain the framework of connective tissue in its simplest and most perspicuous arrangement; it is composed of the neuroglia with the vessels inclosed by it, contains a few multipolar ganglion-cells, and embraces in its meshes the nerve-fibres, sometimes singly, sometimes grouped in bundles of several (Fig. 3).

The large majority of these fibres runs parallel to the long axis of the cord, for which reason they almost always present their well-known cross-section, in transverse sections of the cord. But the longitudinal bundles by no means always maintain an exactly parallel direction among themselves; they deviate from it in many ways, cross here and there at acute angles, often weave into each other, or are seen to change their place in the cross-section by degrees, moving towards the centre or the circumference of the cord, forward or backward.

A certain number of fibres, however, run across the white columns in a more or less exactly horizontal direction. Such are especially the entering fibres of the roots, which run in broad bundles at the level of the section, or only a little off from it. They pass more or less directly from the cortex to the gray pillars [cornua]. The anterior roots mostly reach the gray anterior pillars in the shortest and straightest path, while the bundles of the posterior roots are much interlaced after entering the cord, and pass through a devious and irregular course before reaching the gray posterior pillars. The fibres of the white commissure are horizontal.

Finally, there are a few oblique fibres and bundles in the white columns. They are partly root-fibres, which do not take a directly horizontal course to the gray substance, but first pass obliquely upward or downward in the white substance for a certain distance, and then bend to enter the gray; and partly fibres which, leaving the gray matter in a horizontal direction, change to a perpendicular. This arrangement is especially frequent at the surface of contact between the lateral columns and the gray matter, where an abundance of fibres leave the gray and turn upward or downward in the lateral columns. It is, however, doubtful (although recently reaffirmed) whether root-fibres run *directly* up or downward in the white columns, without first having passed the gray substance; this is affirmed of some bundles of fibres in the posterior roots.

The structure of the gray columns [cornua] is considerably more complicated, and has not yet been examined in at all a satisfactory way. Two kinds of gray matter are usually distinguished in it, plainly recognizable and separable by the naked eye, namely, the *spongy* and the *gelatinous* substance. Their distribution upon the surface of a cross-section is very unequal. The spongy substance composes the chief mass of the gray columns, while the gelatinous substance forms only a semilunar cap over the tips of the posterior cornua, of greater or less thickness (substance of Rolando), and surrounds the central canal in a moderately thick layer. A prismatic column, situated at the boundary between the anterior and posterior cornua laterally from the posterior commissure, and close to the apex of the white posterior column, has been considered by many entitled to rank as a special formation in the cord; it is the *columna vesicularis*, now best known as *Clarke's column* (Fig. 2, *s*). This formation, rich in ganglion-cells, is found only in the dorsal part of the cord, beginning at the upper end of the lumbar enlargement and ending at the lower part of the cervical enlargement.

Careful examination shows that probably the largest part of the *gelatinous* substance belongs to the neuroglia. It consists of the finely granular substance before described, which, however, in this situation is traversed by a few of the finest fibres of connective tissue, and contains a remarkable number of nuclei (glia-cells). It is traversed by many bundles of fine nerve-fibres, running in gentle curves from behind forward in various courses. These bundles originate in part from the posterior root-bundles, in part from the posterior columns, and probably also from the lateral columns. Besides, the gelatinous substance is traversed by vertical bundles, parallel with the long axis of the cord, which are especially distinct in the lumbar cord, and occupy principally the middle and the anterior parts of the gelatinous substance. In them large nerve-cells are rarely found, and the fine network of nerve-fibres discovered by Gerlach is said to be also wanting.

The *spongy* substance is far more complex in structure. It consists of a mixture of fine fibres and bundles of fibres, seemingly inextricable, crossing in all directions, which resolve themselves into fine networks of fibres, only to recompose themselves in the former aspect of bundles of fibres; at the same time they contain numerous multipolar ganglion-cells, arranged in definite groups. The fine nerve-fibres select the spongiosa for the seat of a repeated ramification, and their branches unite to compose a *network of excessive fineness*, discovered by Gerlach. Very similarly, the protoplasm processes of the multipolar cells form with their ramifications a fine network; and it is extremely probable, although not yet certainly proved by direct observation, that the fine bundles of nerve-fibres and the ganglion-cells communicate at numberless points through the medium of this network.

Boll has confirmed Gerlach's discovery in all points, and has added to his statements another, to the effect that this fine network of nerve-fibres is not merely distributed through the entire gray substance, but can also be followed through the septa of the white substance into the gray cortical layer. From this it would follow that this remarkable and important network is universally distributed through the cord.

Schiefferdecker has very recently attempted to follow out with somewhat more minuteness the incredible medley of nerve-bundles in the cord, and to ascertain the course of the principal ones. The most important result of his investigations—and it is hard to apply either physiologically or practically—is that, so to speak, *all* parts of the gray and some parts of the white substance are placed in the most universal and manifold mutual connection by the various networks of nerve-fibre ; the entering nerve-roots form connections with all the groups of ganglion-cells ; the latter are mutually joined by strands of fibres of their own, and send forth bundles of fibres to the white columns ; and all parts of each half of the cord stand in connection with all parts of the other half ; while, finally, higher and lower segments of the cord are joined by vertical fibres.

The spongiosa is crossed in many places by vertical ascending bundles of fibres ; this is especially the case in the neighborhood of the white substance, where bundles detach themselves from the white columns, enter the gray matter, and after a short distance return to the white. This is most developed in the processus reticulares. The longitudinal commissures in the gray substance, just mentioned, appear in transverse sections as vertical bundles of fibres.

The *groups of ganglion-cells* in the gray substance are highly interesting. Their number and mode of distribution in the anterior and posterior cornua vary. They can be seen excellently, with their processes and ramifications, in cross and longitudinal sections. Repeated observations have shown that their nerve-process is directly transformed into a medullated nerve-fibre, and, in the case of the great cells of the anterior cornua, that the fibre passes directly into the anterior roots and mingles with their bundles of fibres ; but this has been shown in the case of but few cells, and in most the nerve-process takes other directions, of which the object is chiefly unknown. It is further demonstrated with certainty that the protoplasm processes of the ganglion-cells break up by repeated ramification into a fine network of nerve-fibres (Gerlach, Boll, Schiefferdecker), and it is probable that this network stands directly connected with the finest nerve-fibres and their ramifications.

Most of the ganglion-cells are found in the anterior cornua; in the cervical and lumbar enlargements, especially, a great number of them are seen in every transverse section. Their distribution is not the same in all parts of the cord, but distinct groups can be made out in most sections. First, a *medial group*, adjacent to the anterior and inner border of the anterior cornua, often broken up into smaller groups; next, a *lateral group*, situated in the anterior external portion of the anterior cornu (Fig. 4, *A*, *a b*), and containing a great many cells; besides which, there is found in the cervical and upper dorsal portion, in the tractus intermedio-lateralis, a third group of very large multipolar cells, which may be designated as the *postero-lateral* group (Fig. 4, *A c*). To the latter corresponds a similarly situated group in the lumbar region; but in this part of the cord the division into sharply defined groups is less distinct, and the cells are distributed more or less irregularly over

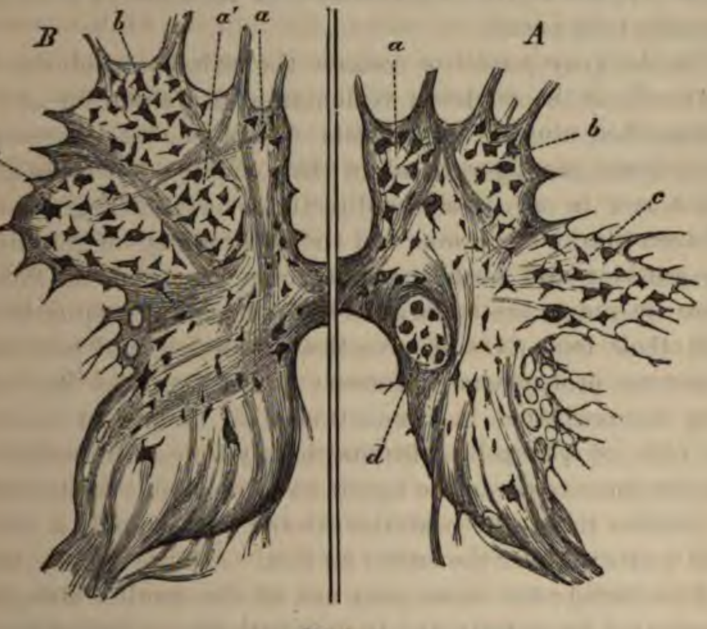

FIG. 4.

Semi-diagramatic transverse sections of the gray substance of the cervical (*A*) and lumbar enlargement (*B*), to show the situation of the ganglion-cells. Magnified $^{12}/_{1}$. *Aa*, medial group; *b*, antero-lateral; *c*, postero-lateral group; *d*, columna vesicularis. *Ba*, medial group; *a'*, group first appearing in the lumbar region, perhaps belonging to the medial; *b*, antero-lateral; *c*, postero-lateral group. In the posterior cornua, only a few scattered ganglion-cells.

the greater part of the section of the anterior cornua (Fig. 4, *B*). The arrangement of these groups of cells in different sections is extremely variable, and the number which can be distinguished is sometimes greater, sometimes less.

All these groups are found throughout the length of the anterior gray matter, forming literal columns of cells. Amongst the groups, and through the whole mass of the anterior cornua, many single ganglion cells with more than one process are found, more or less abundant in different sections. Even in the white columns in the neighborhood of the anterior cornua, a few sporadic cells occur.

In the gray *posterior cornua* the principal column of ganglion cells is the columna vesicularis. Its situation and extent is described above. It consists chiefly of closely-crowded fine nerve-fibres, mostly vertical in their course; but there are fibres which run in all possible directions, establishing connections between Clarke's columns and the other groups of ganglion cells, the anterior and posterior root-fibres, etc. Among these fibres there lie many pretty large nerve-cells, mostly spindle-shaped, with their long axes in a vertical direction, and provided with numerous protoplasm-processes; a nerve-process has not yet been demonstrated in connection with them. In cross-section the cells of Clarke's columns mostly appear roundish. Their size increases towards the upper and lower ends of the cord.

Besides these, the posterior cornua contain only a few nerve-cells scattered over the entire section. A large multipolar cell is seldom found; the most part are of the smaller sizes, and are distributed irregularly and in very variable quantities among the spongy tissue of the posterior cornua. Nerve-processes have not been certainly demonstrated in connection with them; but the part they take in the formation of the fine network of nerve-fibres may be considered as established.

The *central gray substance*, which unites the four gray columns and encloses the central canal, chiefly consists of a finely granular and finely fibrillated mass (neuroglia, gelatinous substance), containing many cell-bodies or nuclei. This substance is traversed by a fine network of nerve-fibres with broad meshes (Gerlach), and also contains bundles of fibres, of greater or less thickness, passing from one-half to the other of the spinal cord, before and behind the central canal, sometimes taking the direct transverse course, and sometimes ascending and descending obliquely (gray fibres of the anterior and posterior commissures).

The *white commissure* consists almost entirely of nerve-fibres with dark contour, most of which run in a horizontal plane, decussate at an acute angle, and pass from one lateral half of the cord to the other, diagonally from front to rear. But a few bundles ascend obliquely, and immediately assume a perpendicular direction. The white commissure first connects the gray columns of one side with the anterior columns (white) of the opposite side (Gerlach), as the bundles of fibres which leave the former pass upward in the latter; it further connects the various cell-groups in the gray columns with one another, and finally, it contains fibres which traverse the gray substance and pass into the white lateral columns.

The *central canal* is a very narrow passage, often obliterated or occluded, presenting a roundish or elliptic, sometimes a triangular transverse section. It is lined with a ciliated cylindrical epithelium, and its wall is formed of dense undulated connective tissue of extremely fine fibrous structure (ependyma). It is bounded externally by the central gray substance, and is filled with a fluid, probably identical with the cerebro-spinal fluid.

Anatomical data concerning the connection between the histological elements, and the course of the fibres in the spinal cord.

Many studies have been made by anatomists, with a view to unravelling the connection between the separate elements of the cord, the course of the entering root-fibres, their connection with other fibres, and with the ganglion cells, and finally, their ultimate destination, or their continuation to the brain. Unspeakable trouble and toil have been devoted to these examinations; absurdly small and trifling have been the results; hardly anything is established with certainty, and the liveliest controversies are still kept up upon almost all points. But every fresh examination reveals fresh complications of the course of the fibres, which reduce the attempt to disentangle and follow them out almost to an impossibility; and the deeper we go into the finer structural relations of the cord, the plainer it becomes that a separation and isolation of the individual groups of fibres or cells is not what is sought or reached, but rather a connection among them, as universal and complete as possible. This naturally renders it extremely hard to reach the desired goal.

We shall here attempt to state, as briefly as possible, what may at present be regarded as somewhat certain, and likewise, what may be regarded as at least probable. For all details and further explanations we refer to the works of Stilling, Clarke, Koelliker, Frommann, Gerlach, Deiters, Goll, Henle, Boll, Schiefferdecker, and others.

The following points seem at present pretty well established:

That all, or at least by far the greater part, of the nerve-roots pass straight to the gray substance, and enter it; this is certainly true of the anterior roots, but perhaps not of some small portion of the posterior root-fibres.

That a large number of these entering root-fibres unite with the ganglion cells or their processes; this also seems quite certain for the anterior roots, but is still doubtful in respect to the posterior.

That numerous fibres pass from the gray substance into the antero-lateral columns, especially the lateral columns, bend upwards, and in the white columns take the direction towards the brain. Some of these nerve-fibres, leaving the anterior gray columns, pass into the white commissure and thence into the anterior white column of the other side of the cord (decussation within the cord), in which they probably run to the brain. Others pass into the lateral columns, in which they run up into the medulla oblongata, and finally decussate in the pyramids.

That numerous fibres from the posterior gray columns enter the posterior white columns (and the posterior regions of the lateral columns ?) where they bend upwards and run further in the direction of the brain.

That the ganglion cells of the gray substance are mutually connected by numerous processes in the most complicated manner, both in the anterior and posterior cornua of each lateral half, and by means of the commissures which join the two lateral halves with each other; that moreover, processes of these ganglion cells pass, some directly into the root-fibres, and some into the white columns, where they assume a vertical direction.

That of the fibres which enter with the posterior roots, a part pass horizontally directly forward, and lose themselves in the fine network of nerve-fibres, or reach the ganglion-cells of the anterior cornua; while another part runs upward or downward at the side of the posterior gray column, then bends again, and at last enters the gray substance.

That the fine nervous network, and the nerve-bundles which by their resolution compose it, and the bundles which are recomposed from it, serve to connect the groups of ganglion cells in all possible directions, with each other, with the entering root-fibres, with the white columns of both sides in the sagittal, transverse and vertical directions.

The following statements may be regarded as more or less *probable*, but not at present certainly proved:

That after the root-fibres enter into certain ganglion cells, processes from *the same* cells pass directly into the white substance (going from the anterior cornu into the antero-lateral column, from the posterior cornu into the posterior column and the posterior part of the lateral column); in the white substance they ascend directly to the brain.

That individual fibres of the anterior and the posterior roots meet each other in certain cells of the gray substance.

That a few bundles from the anterior roots merely traverse the gray substance, going thence directly into the anterior parts of the lateral column, and there bending upward. Their significance is not yet clear.

That the posterior root-fibres first enter into the fine network of nerve-fibres, in the gray posterior columns, and that the connection with the ganglion cells is accomplished through this network (Gerlach).

That each single ganglion cell, by means of its branching processes, passes into a fine network of nervous fibres, from which larger medullated fibres are again developed (connection of the cells with the nerve-fibres).

That the network of nerve-fibres into which the posterior root-fibres break up is in continuous communication with the network of nerve-fibres in the anterior gray columns; that from this latter network numerous fibres originate, which cross the median plane in the gray commissure, and then ascend toward the brain, some in the vertical bundles of the posterior cornua, some in the posterior columns (total sensory decussation in the cord ?). The gray substance, therefore, seems to be much more intimately connected with the posterior nerve-tracts (through the fine network) than with the tracts which prolong the anterior nerve-roots.

That from Clarke's columns bundles of fibres pass out into the lateral columns.

That the medial parts of the posterior columns (so-called fasciculi graciles) have by their development and structure a special significance, which is at present entirely obscure. Pierret considers them as a great longitudinal commissure, intended to bring various parts of the gray substance into mutual connection.

All these results are defective and inadequate in the highest degree; they hardly permit us to form an exact idea of the complicated course of the fibres. In general, we can only infer from all this that the root-fibres which pass from the peripheral nerves into the cord first enter the gray substance, where they have their first termination; that they next, after many ramifications and connections, leave the gray substance and pass up the white columns to the medulla oblongata. This process is repeated step by step at the entrance of every new pair of roots.

But this scanty result gives us no information regarding the precise direction of the single fibres, none regarding their connections, none regarding the physiological significance of the several groups of fibres. Anatomy can at present give us no certain information upon these points; we must expect it rather from the most scrutinizing *physiological research*. The next section will be devoted to explaining the paths of conduction in the cord, as at present understood through the aid of physiology and the equally important aid of pathological anatomy.

Let it only be remarked in this place, that the anatomical formation of the cord, which we have just attempted to sketch, is somewhat modified in its uppermost region by the accession of some new parts. We refer to the *origin of the accessorius*, whose roots are seen leaving the lateral column as far down as the fifth and sixth cervical vertebræ. Within the cord they can be followed to the ganglion cells of the postero-lateral cell group of the anterior cornu.

In analogy with this, the so-called *ascending root of the trigeminus* can be followed in the cervical cord to about the level of the third cervical vertebra in the form of a large bundle of fibres, which is in relation with the substantia gelatinosa of the posterior cornu, and gradually passes into it.

Finally, in the uppermost part of the cord, *the decussation of the pyramids* produces a sort of substantia reticularis—a manifold interweaving of the bundles of fibres—which is visible in the middle of the anterior half of the cord.

Concerning the further course in the medulla oblongata of the nerve-paths which leave the spinal cord, see further on, under the proper heading.

II. Physiological Introduction.

Cf. Longet, Anatomie et physiologie du système nerveux; German transl. by Hein, 1847.—*Schiff*, Lehrb. der Physiol. des Nervensystems. Lahr, 1858–59.—Centralbl. f. d. med. Wissensch. 1872. No. 49.—*Brown-Séquard*, Experim. and clinical researches on the physiol. of the spinal cord, etc. Richmond, 1855. Course of lectures on the physiol. and pathol. of the central nervous system. Philadelphia, 1860.—*Sanders*, Geleidingsbanen in het ruggemerg. Groningen, 1866.—*Wundt*, Physiologie, 1873. 3. Aufl.; Physiol. Psychologie. Leipzig, 1874. —*Hermann*, Grundriss der Physiologie. 2. Aufl. 1874.—*Leyden*, Klinik der Rückenmarkskrankheiten. I. 1874.—Also innumerable articles in Reichert and du Bois-Reymond's Archiv, Virchow's Archiv, Pflüger's Archiv der Physiologie, Zeitschr. f. wissensch. Zoologie, Zeitschr. f. rationelle Medicin, Moleschott's Unters. z. Naturlehre, the Monatsberichte of the Saxon Academy, Brown-Séquard's Journal de la physiol. de l'homme, etc., the Archives de la physiol. norm. et pathol., etc.

The simple fact that traumatic destruction of the cord at any place, or its experimental section, completely suspends the sensitive, motor, and vaso-motor connection between the brain and the periphery of the body, was sufficient to prove that the channels for maintaining this connection lie in the spinal cord; in making a step beyond this it became necessary to employ a great variety of experiments to establish with accuracy the course and the situation of these various channels. In order to accomplish this, physiology has made colossal efforts, and has produced a series of most valuable studies, incomplete though they be in many respects. Of late years, pathology has contributed not a little to enlarge our knowledge of the physiology of the cord.

Unfortunately, the physiological as well as the pathological methods of investigation, and especially those of pathological anatomy, are still very imperfect. In the experiments by section, which are the most frequently employed, the effect of the primary shock and of the secondary inflammation are hard to separate from that of the simple severing of the channels of conduction; secondary degeneration also frequently occurs, and disturbs the result of the experiment. This manifestly gives rise to uncertainty and confusion in the statement of results, which is increased by the difficulty of obtaining from animals exact objective reports of the disturbances of function. In man, on the contrary, it is comparatively easy to ascertain in pathological cases the nature, the degree, and the extent of the disturbance of function; but it is less easy, and in fact, quite a matter of chance, to obtain for anatomical investigation exactly the right stage of the disease, and it is harder still to find the histological changes defined exactly and beyond a question. This must always be borne in mind, that we may not suffer ourselves to repose in too great confidence of the value of our knowledge.

In the following paragraphs we shall attempt to present a brief summary of what is established in the physiology of the cord, or at least, that which is probable, and capable of being used by the pathology of our day.

Sensory Conduction in the Spinal Cord.

All the impressions received by peripheral sensitive nerves[1] are conducted to the spinal cord by the posterior roots; they pass first into the gray substance, and thence are conducted into the posterior columns and a part of the lateral columns, in which they ascend to the brain.

The principal channel for sensations of touch, pressure, temperature, tickling and the like, is to be sought in the white posterior columns.

Section of the white posterior columns destroys the sensation of touch permanently in the regions situated posteriorly to the section (Schiff); but it does not annihilate every sensation, as a hyperæsthesia, especially in respect to painful impressions, continues for a time, gradually disappearing. It is not yet determined whether there exist separate paths of conduction for the different varieties of the sense of touch, but Brown-Séquard maintains this view and supports it by weighty pathological facts; he supposes these paths to lie mostly in the gray substance. The latest experiments of Woroschiloff (in Ludwig's laboratory) are of great interest; they seem to show that the lateral columns are of far more importance in conducting sensation than has hitherto been supposed. But these experiments are confined to the lumbar cord of the rabbit, and cannot yet be made the basis of more general conclusions. They appear to show that *each* lateral column contains sensory fibres for *both* legs; the more important seem to *decussate.*

The sensation of pain is conducted chiefly or exclusively by the gray substance.

Schiff makes section of the posterior columns destroy the sense of touch, but not that of pain; while section of the entire gray substance, leaving the posterior columns intact, destroys the sense of pain, and leaves that of touch (the condition known in pathology as *analgesia*).

The presence of the network of nerve-fibres shown by histology, hardly permits us to explain these facts by the assumption of separate paths of conduction; hence Wundt (Physiol. Psychologie, p. 117) has proposed the hypothesis of different excitability of white and gray matter; the gray requires, in order to produce a reaction, a much higher and more intense irritation than the white; but when the reaction occurs, its intensity is all the greater, and produces pain. If, therefore, the gray substance alone remains for the purposes of conduction, more powerful irritations are usually required; and when sensation is produced it is more violent—that is, painful; but if the white columns alone are retained, the irritation quickly reaches a degree at which sensation is produced, but never goes so far as to produce pain.

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[1] According to Brown-Séquard, the paths for the "muscular sense" lie in the anterior roots.

The gray matter conducts sensation even after the section of all the white columns; it does this in its entire section and in every part of it, although it is quite inexcitable under direct irritation, and is, therefore, designated as *æsthesodic* substance (Schiff). In this sense the white substance, with the exception of the posterior root-fibres which traverse it, was supposed to be also æsthesodic; the experiments of Engelken, Fick, and Dittmar, however, seem to have finally settled that the paths of sensation which have once passed through the gray substance of the cord are still excitable.

The anterior and the greater part of the lateral columns have nothing whatever to do with the conduction of impressions of sensation.

The *conduction of sensory impressions decussates* in the cord soon after the root-fibres enter it; this decussation seems to be pretty complete in the dorsal and cervical medulla of man (Brown-Séquard, Schiff). The decussation of the sensitive paths is certainly complete in the medulla oblongata.

Whether the paths of all the kinds of sensation decussate is not yet fully determined; according to Schiff's later statements those for the sense of touch do not; according to Brown-Séquard, those for the muscular sense also do not. The latter author also says that the channels for the various kinds of sensation decussate at different heights. Miescher found decussation of the centripetal (sensory) fibres from the sciatic, which produce a reflex increase of the blood-pressure. The fact of the sensory decussation in the cord is established beyond any reasonable doubt by numerous pathological observations.

The isolated conduction of the separate sensory impressions can only be explained (in view of the fine network) by supposing that certain tracks in the conductive substance offer less resistance than others, and are, therefore, habitually employed. Such tracks are probably constituted by the fibres which pass directly from the network of nerve-fibres into the posterior (lateral) columns and pass upward in the latter to the brain; these, under normal circumstances, offer least resistance. The further extension of powerful sensory impressions, or the conduction which continues to take place after interruption of the principal channel, is readily explained by reference to the network.

In the same way is explained the transference of powerful irritation to neighboring or distant sensitive tracks, producing the associated sensations; they require only a diminution of resistance in certain channels, or an increase in the strength of the irritation.

A retardation of the conduction of sensation occurs when the posterior columns are entirely cut, and only a part of the gray substance remains; the more the gray substance is diminished, the more distinct is this retardation (Schiff); this fact may very well be used to explain that retardation of the conduction of pain which not infrequently occurs in pathological cases.

In regard to the position of definite sensory paths in the cord, physiology informs us that in the case of the lower extremities these paths lie at first in the lateral columns, and do not enter the posterior columns till a higher point; the posterior columns of the lumbar cord are said to contain only the nerves of touch for the pelvic region, sexual organs, perineum, and anal region.

Motor Conduction in the Spinal Cord.

This subject has by no means been examined in all points. The principal line of conduction for voluntary movements passes from the brain into the cord, through the decussation at the pyramids, and probably to a still greater extent through other routes of decussation in the medulla oblongata and pons. The motor (voluntary) paths do not further decussate in the cord, but remain upon that side which belongs to the half of the body destined to receive the nervous influence.

Most of the paths for voluntary motion probably run down in the lateral columns, enter the gray substance at different heights, form connections, through the network of nerve-fibres, with the large multipolar ganglion cells, and pass through their axis-cylinder processes into the anterior roots. The fibres for voluntary motion all lie in the anterior roots; these roots, however, contain other fibres which possess a different physiological function.

Section of the posterior columns and the entire gray substance does not destroy the voluntary movements of the portion of body lying behind the point of section. Section of the antero-lateral columns and the entire gray substance destroys all voluntary motion in the corresponding parts. Section of the anterior and the lateral column lessens voluntary motion for only a short time, and motion returns the quicker, in proportion as the gray substance is retained. In regard to the function of the anterior columns proper we are still greatly in doubt; they do not seem to aid in voluntary motion; they are supposed to carry fibres, for the most part, whose function is to transmit reflex impulses originating in the brain (Huguenin); they also bear fibres which experience one more decussation in the cord, and pass through the anterior commissure into the gray anterior column of the other side. In these points, also, Woroschiloff's experiments (upon the lumbar cord) have brought new and unexpected facts to light, which ought, however, to be applied with extreme caution. Motor baths for *both* legs are contained in *each* lateral column; the more important of these, those which produce reflex action, co-ordination, etc., seem not to decussate.

Even after section of the antero-lateral columns, a translation of motor impulses to the posterior half of the body is possible through the gray substance, and even through certain limited portions of it. This substance is inexcitable under a great variety of stimuli, at the same time that it conducts the impulses of motion; it is therefore *kinesodic* (Schiff). This is by no means true of the longitudinal fibres of the antero-lateral column; they are not kinesodic, as is affirmed by many physiologists, who seek to derive all the phenomena of motion which occur when the antero-lateral columns are irritated, from irritation of the root-fibres which traverse them; the experiments of Engelken and Fick leave not the slightest doubt that the anterior (lateral ?) columns of the cord are as truly excitable as any other nerve-fibre.

The isolated conduction of individual motor impulses, like that of the sensory excitations, is explained by the supposition that of the many channels which are open some offer less resistance, and therefore are usually selected. But even in this region many translations to other tracts (associated movements) occur, either because the normal paths are not sufficiently used, or because the resistance offered by other paths is diminished, or because the irritation is increased in force.

As respects the position of certain motor paths in the cord, we will here mention the statements of Schiff, that the lateral columns of the upper cervical portion contain those for the muscles of respiration, and that cutting them destroys *permanently* the movements of respiration on the same side. But this is denied by others. Woroschiloff found in the lumbar cord of the rabbit the motor paths for the foot and leg below the knee lying towards the outer circumference of the lateral column, and those for the thigh more towards the middle.

Co-ordination of Movements.

The spinal cord plays a considerable part in this important function, and disturbances of co-ordination of movement are not at all rare in spinal disease; for these reasons, and in view of the numerous unsettled controversies connected with the matter, we feel ourselves bound to give a careful statement of the difficulties which surround it.

What is meant by co-ordination of movement, is not difficult to define; it consists in *the innervation of a large number of muscles simultaneously, each with a different but appropriate degree of force, for the purpose of attaining a given object of motion.*

A little close inspection will reveal the fact that muscular motions, even those which look simple, as lifting a burden or throwing a stone, are really quite complex, and involve a large number of muscles; still more is this the case in the more complicated acts of writing, piano-playing, gymnastic feats, and the like.

The manner in which this wonderful mechanism regulates the co-ordination of movements, and the methods it takes to accomplish its results, can best be seen by observing children or persons who are learning to perform any complex motion, as writing or piano-playing.

In the new-born child but few co-ordinate acts seem prepared for; the movements of respiration, of sucking, crying, and swallowing, and perhaps those of the eyes, are performed immediately. All other co-ordinated motions must be learnt with pains and by a thousand attempts, as is the case with standing, walking, running, and especially speaking, and later, writing and all sorts of skill in the use of the hands. The apparatus for co-ordination seems to be ready formed, but not to be in readiness for full use ; it attains full development by use and varied practice. It is conceivable and probable that the frequent use of the nervous paths (in the fine network?) in certain directions by degrees overcomes the resistance along these lines, until at last their employment becomes almost a matter of course.

The process which takes place when co-ordinated movements are being learnt, may be somewhat as follows: The will sends down an impulse, and the part of the central apparatus which it first reaches is that which presides over the association and co-ordination of the single impulses of motion. This takes place under the constant supervision of the sight (in the case of speech, of hearing also), and of the peripheral sensations of the skin and muscles ; which convey a conscious impression of the correctness or inaccuracy of the movements, and enable the person to apply to them the proper corrections. By continued exercise and repetition the movements become more and more perfect, and may thus reach a high degree of precision.

When co-ordination has once been acquired, and the motor paths concerned have been sufficiently trodden, the complicated movements take place quite automatically, in response to a simple impulse of the will, aided by the apparatus for co-ordination. A supervision by the sight or the sense of touch is then no longer needed. This clearly appears from the fact, that after a little practice we can execute the most complicated motions with a swiftness and sureness which prove that they are not at all controlled by any active and defining regulative sensation—as when we grasp at a certain object, throw at a mark, leap over a ditch, play the piano in the most rapid time, etc. We therefore are able so to determine in the central organ the arrangement and force of the various processes of innervation, and by the aid of the will and the co-ordinative apparatus so to bring them to pass, that a completely ordered movement is the result. At any rate, this is the way in which most co-ordinate movements are performed, when once they have been sufficiently practised and learnt, as walking, running, grasping, writing, speaking, etc.

The question as to the anatomical seat of the centres of co-ordination and the centrifugal paths belonging to it, is not completely settled. According to the latest researches (among which those of Goltz are of especial importance) it appears that the proper centres of co-ordination lie *in the brain only*. The corpora quadrigemina, thalami optici, and cerebellum are the organs which seem to take the most prominent part in the co-ordination of movement.

In the spinal cord there seem to be no such centres, although the undoubted fact that orderly reflex movements may be evoked from the cord is sufficient proof that combined movements, serving definite purposes, can be arranged in the cord. But the point we are here interested in is the co-ordination of voluntary movements, which has little to do with the apparatus for these reflex actions.

It seems, upon the whole, that *the spinal cord contains only those paths of conduction which lead the co-ordinative impulses to the muscles*, which, therefore, place the cerebral centres of co-ordination in connection with the anterior roots.

In what part of the cord these co-ordinative paths lie, and in what manner they enter into connection with the motor paths, is at present wholly unknown. Pathological facts, to which we shall come later, permit us to suspect that these paths are to be sought in the white posterior columns or in their immediate neighborhood; and in order to supply the connection with the various nerve paths the fine network of nerve-fibres may also be called in play. But the physiological researches of Woroschiloff—which, however, refer only to the lumbar medulla—show that the co-ordinatory paths lie in the middle third of the lateral columns, in the hollow between the anterior and posterior cornua. This agrees with a statement by Schiff, that the symptoms of ataxia may be produced in the lumbar cord by lesion of the lateral columns.

In the closest connection with the co-ordination of movements we may here speak briefly of the processes which enable us to *retain the equilibrium of the body*. In the execution of this function a large number of accurately and finely co-ordinated muscular contractions are concerned, which continually alter the centre of gravity of the body in such a way that its equipoise is kept and the body remains upright. For this purpose a constant oversight by the senses appears to be necessary, to inform us of the position of the body in space and of the posture and position of the parts of the body. The oversight is exercised partly by the sensibility of the soles of the feet, the joints, the muscles, the skin, etc., partly by the sense of sight, and perhaps also by the semicircular canals of the labyrinth of the ear. These centripetal stimuli, constantly in activity, are converted in the central organ into definite co-ordinated movements, which preserve the equilibrium of the body. The co-ordinating centre which preserves the balance of the body is supposed to lie in the corpora quadrigemina and the thalami optici. The conductive paths appertaining to it lie in the spinal cord, of course, with the exception of those which come from the organs of sight and hearing. The sensory paths for this function are situated, without doubt, in the posterior columns and the gray substance; the situation of the centrifugal paths is unknown.

Vaso-motor Paths and Centres in the Spinal Cord.

These have been the object of much and repeated investigation down to the most recent time. Cutting the cord at any point produces a transitory but very considerable dilatation of all the arteries below the point of section; irritation of the cord, on the contrary, produces a contraction of the arteries below the point of irritation. From this we may infer that vaso-motor paths run in a centrifugal direction in the cord. They are said to be chiefly contained in the lateral columns, and in part, probably in the gray matter also; they are supposed to decussate for certain parts of the body, especially for the vessels of the thigh and trunk (Schiff). This statement, however, is denied (von Bezold).

The centres of vaso-motor innervation certainly lie in the cord and medulla oblongata. It has hitherto been generally assumed that the chief centre lies in the medulla oblongata; but the researches of Goltz, Schlesinger, Vulpian, and Mor. Nussbaum have now established beyond a doubt that vaso-motor centres are found throughout the length of the cord as far down as the lumbar region. It is from these centres that the tone of the vessels is re-established when they have been dilated after section of the cord. The wound at first inflicts a shock, with temporary paralysis; hence the dilation which directly follows the section. As soon as the centres have recovered, the vessels return to their normal volume; every fresh section of the cord at a lower point produces the same series of phenomena. A continued, or even a permanent paralysis of the vessels may occur, but only when the entire cord is destroyed; in this case the irritation of peripheral sensory nerves no longer produces a reflex contraction or dilatation of vessels, in the way observed when the lumbar cord, including the vaso-motor centres, is intact.

But even when the lumbar cord is totally destroyed, the vessels of the posterior half of the body are neither permanently nor wholly paralyzed; the original dilatation gradually diminishes, and the cutaneous temperature, at first considerably increased, sinks again to the normal point or beneath it. The same is the case after section of the sciatic nerve. These facts have compelled us to suppose that the vessels also possess a peripheral gangliar apparatus, like that of the heart, which preserves their tone and keeps them at a certain degree of dilatation, even when they are cut off from all connection with the nerve-centres.

Goltz[1] has recently attempted to refer all the vaso-motor phenomena which occur during the various experiments in section of the nerve to irritation of *vaso-dilator* nerves. These, when irritated by the cut, he supposed to act upon the peripheral ganglia like a sort of nerve of arrest, paralyzing their activity, and thus producing a lax condition of the vessels. According to this view the cord contains only that class of vaso-motor centres which cause dilatation of vessels. In spite of the elaborate defence of this position—which is also held by Vulpian—it has not proved sound, and a further series of researches, made by Putzeys and Tarchanoff in the laboratory of Goltz, has again shaken the theory of vaso-dilator nerves.[2] They refer the symptoms to a great exhaustibility and an excessive stimulation of the vaso-motor paths, which result in immediate dilation of the vessels, preceded regularly by a brief period of contraction.

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[1] *Pflueger's* Archiv. Bd. IX. S. 174.

[2] Centralbl. f. d. med. Wissens. 1874. No. 41.

The vascular tonus, therefore, is under the influence of certain very complicated sets of apparatus; those of the peripheral nerves come first, but are subordinated to the centres in the spinal cord, so that when the latter are excluded, the former require some time to develop their entire activity and to restore the tone of the vessels. This increase of the activity of the peripheral ganglionic apparatus is perhaps favored by the increased influx of blood which occurs after the spinal centres have been excluded. A similar relation may exist between the spinal centres and the vaso-motor centres in the medulla oblongata.

It is not known where the vaso-motor centres in the spinal cord are situated: probably in the gray anterior pillars. The vaso-motor nerves which come from these centres mostly lie in the lateral columns; they leave the cord in the anterior roots; those destined for the head come from the cervical cord, those for the upper extremities from the upper dorsal, those for the pelvis and lower extremities from the lower dorsal and the lumbar cord; the abdominal viscera receive their vaso-motor nerves through the splanchnic, and the uro-genital apparatus from the lumbar nerves.

Trophic Centres and Paths in the Spinal Cord.

Physiology is entirely in doubt respecting the existence and the mode of action of trophic nerves. Pathological facts, in numbers, have continually pointed to the existence of some such trophic influence, coming from the nervous centres; but no generally accepted basis for a doctrine of the trophic nerves and their functions has ever been established. We may therefore confine ourselves to a few remarks, and avoid a close discussion of this section of the general pathology of the spinal cord.

The influence of the nervous system upon the processes of secretion is probably no longer doubted, in view of what we really know concerning the secretion of saliva. It is palpable that these processes have the very closest analogy with processes of nutrition. That the nutrition of most of the peripheral parts, the nerves, muscles, bones, joints, skin, hair, nails, etc., depends in many respects upon the spinal cord seems to follow from numerous pathological facts which have been collected by Charcot.[1] These observations show that many and various trophic disturbances—both those of an inflammatory and gangrenous nature, and also simple atrophy and degeneration—occur in all the parts named when their nervous connection with the spinal cord is cut off, or when the latter itself is affected in certain ways and in certain regions.

The nature of these trophic influences and the routes which conduct them are in most points problematic. The proper centres for these influences are probably the ganglion-cells, especially those of the gray anterior pillars. The routes over which the trophic influences are conveyed to the periphery run in the motor and sensory nerves; but it is questionable whether there exist for the purpose special trophic nerve-fibres, or whether the motor and sensory fibres themselves assist in conveying trophic influences. At all events, no special trophic nerves are anatomically demonstrated at present.

Respecting the position of the trophic centres for special tissues, something is known, but most is obscure. Those for sensitive nerves seem to lie in the spinal ganglia, as found by Waller and confirmed by Schiff. There are numerous and well-established pathological facts in favor of this view, as degeneration of the posterior roots, with unimpaired nutrition of the peripheral sensitive nerves, in cases by Charcot, Vulpian, Schueppel, and others.

The trophic centres for motor nerves and muscles doubtless lie in the anterior cornua, and are usually supposed to exist in the large multipolar ganglion-cells. In the same situation, according to pathological facts, the centres of nutrition for bone and joints are probably to be found (see Infantile Spinal Paralysis). But the centres of nutrition for the skin and its adnexa are probably elsewhere; they are apparently to be sought in the central gray substance or the posterior columns, their nerve-fibres leaving the cord with the posterior roots; perhaps they exist in the spinal ganglia also.

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[1] Clinical Lectures on Diseases of the Nervous System.

Further research is required in order to determine all this. The experiments of Eichhorst and Naunyn[1] have lately shown that the spinal cord contains the means of self-maintenance and self-nutrition.

Reflex Action of the Spinal Cord.

The production of reflex movements—*i. e.*, the direct transference of sensory excitation to motor paths, unassisted by the intelligence and the will—may be assigned without contradiction to the *gray substance.* All spinal reflex acts—*i. e.*, all reflex acts which occur after the brain has been severed from the spinal cord—require, without doubt, the aid of the gray substance of the cord to produce them. These results are confirmed by a great variety of physiological experiments and by numberless pathological facts.

In spite of this, we are not yet entirely clear in regard to the reflex apparatus, and the precise course taken by the excitation which produces the reflex action; yet it is pretty certain that ganglion-cells constitute the proper apparatus for producing reflex action, and that it is in them that the transference of centripetal sensory excitation to centrifugal motor paths takes place; experiments show, further, that the entering root-fibres must connect with ganglion-cells very soon after passing into the gray substance.

The centripetal paths, which convey a stimulus inward, lie beyond a doubt in the posterior roots; those which convey forth a stimulus, the centrifugal, or motor, lie in the anterior roots; but of that which lies between these two routes, and its histological structure, we are not well informed. We may, however, guess that there are branch conductors given off both from the sensory and from the motor paths at various points within the spinal cord, which meet each other at certain ganglia and groups of ganglia (reflex centres), and enter into conductive communication with each other; but these conductors may be supposed to be connected by means of the fine network of nerves with all other possible paths in the gray matter, up to a great distance from their proper seat, so that a reflex motor excitation, originating from a single point, may be diffused more or less extensively. The reflex excitation *may* therefore pass to many or even all of the motor paths; but, as a rule, it passes to but a few, and often to but a single one.

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[1] Arch. f. experiment. Path. u. Pharmak. II. p. 242.

There exist numberless paths, with very various resistances to conduction; those which present the least resistance are first occupied. If the strength of the irritation is increased, or the resistance within the reflex paths is diminished, the reflex movements are correspondingly increased in extent.

In harmony with this complication is the fact, that the time required to carry out the reflex conduction is many times (according to Helmholtz 11–14 times) greater than that required for simple motor conduction.

The degree of reflex irritability differs very much in different persons; in many, all possible reflex acts can be produced with the greatest ease, while in others this is very difficult, or impossible. Various physiological conditions, many poisons, and especially pathological conditions, have the power to modify the reflex irritability to a considerable extent.

The first and ordinary consequence of a brief sensory irritation is a simple brief contraction of the muscles, or a more prolonged tetanic contraction; subsequently repeated convulsive jerks also occur; Freusberg[1] and Goltz[2] have lately observed reflex actions that intermitted rhythmically, following a single or continued irritation; in the higher degrees of excitation the muscular contractions become more and more extended, and almost the entire musculature may at last take part in the reflex action, as occurs in many forms of general spasm.

The way in which the reflected actions increase in extent, while the irritation is increased, has been carefully studied by Pflueger, who found the following results: The excitation, passing from a sensory fibre, is first transferred to motor fibres on the same side and the same level of the cord; next it passes to the symmetrically situated fibres of the other side, but in a somewhat weaker degree; then motor fibres in other sections of the cord are attacked, first those lying above toward the medulla oblongata, and afterwards those situated lower down; finally, general reflex contractions of the great part of the muscular system occur.

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[1] Reflexbewegungen beim Hunde. Pflueger's Archiv. IX. p. 358.

[2] Ueber die Functionen des Lendenmarks des Hundes. Ibid. VIII. p. 460.

The reflex actions are not always simple movements; more or less complicated movements occur, which may even have the appearance of adaptation to a certain end (movements of self-defence, of flight, and the like), in which cases there doubtless exists a simultaneous excitation of several motor paths, which are associated in the cord for certain purposes, or are united by habit. There are, however, actual series of motions, motions with a proper sequence, which serve a given purpose; such are, for instance, the rhythmic twitching of the hind legs, described by Freusberg, the processes concerned in defecation, etc. These cases are sometimes instances of a stimulus to new motion, originating in the first reflex action; or they are cases of the stimulation of entire centres, which govern various physiological acts at once.

Reflex actions may be originated by stimulus applied to any sensitive part of the body. Cutaneous reflex actions are the best known, originating in stimulation of the skin; the most sensitive regions in this respect are the sole of the foot, the face, the front of the belly, the inner surface of the thigh. Irritation of the skin excites in different persons more or less generalized reflex actions, which are strictly obedient to Pflueger's laws of reflexion.

The tendinous reflexions, lately described by Westphal[1] and myself,[2] are of great importance in pathology. The tendon of the quadriceps and the ligamentum patellæ, the tendo-Achillis, and the triceps tendon in the upper arm, are the best points for demonstrating these reflex actions, as yet only observed in man. They are caused only by mechanical irritation (light tapping with the finger or the percussion hammer), are very easily distinguished from the cutaneous reflex actions, and are strictly confined to the muscles and groups of muscles belonging to these tendons. Similar reflex actions can be originated, at least in cases of pathological increase of excitability, from the periosteum of many bones, from fasciæ and articular ligaments.

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[1] Ueber einige Bewegungserscheinungen an gelähmten Gliedern. Arch. für Psych. u. Nervenkrankh. V. p. 803. 1875.

[2] Ueb. Sehnenreflexe bei Gesunden und bei R.-M.-Kranken. Ibid. V. p. 792.

Westphal's article, containing a great quantity of interesting and valuable material bearing on the phenomena in question, appeared after I had written the above. He gives the name of "lower leg phenomena" to that which I denominated the tendo-patellæ reflex action, and the name of "foot phenomenon" to the reflex clonus which occurs when the foot is passively moved in dorsal flexion (to be described in the section on general symptomatology under "Increase in Reflex Activity"). The article shows that Westphal does not consider these as reflex acts, but believes that the muscular contractions are produced *directly* by mechanical stretching and shock of the muscular substance. The fact that this is most easily produced at the tendon depends on the facility with which the fibres of the muscle can be mechanically irritated *all at once*, by pulling the tendon. Westphal, therefore, considers the phenomenon as due to a direct irritation of muscle, and compares it in pathological cases with abnormal states of muscular tension and contraction.

We ought, I think, to have very convincing grounds for abandoning the theory which lies next at hand, with its numerous physiological analogies, especially when the positive reasons for the alternative theory are so very few. I can by no means admit the existence of such reasons, as opposed to the reflex theory of these phenomena. Moreover, a great number of positive facts, which I have since collected, and which can easily be proved, even upon many well persons, are so decidedly in favor of the reflex theory, and against the theory of direct muscular irritation, that all my doubts are completely put to rest. I will mention only a few of these briefly. In many patients, the quadriceps reflex action can be produced by moderate tapping on a large part of the free surface of the tibia. Tapping on tendons in places where firm substance underlies them (*e. g.*, tendon of the tibialis posticus under the malleolus) produces the reflex action. In both these cases, all mechanical action upon the muscle is avoided. We can produce the effect upon the biceps femoris (in patients) by taking up the tendon in our fingers, in an entirely relaxed condition, and pinching it a little while; this succeeds, even if the tendon just above the pinched part is held firmly with the other hand. The reflex action of the supinator longus can be produced from the lower end of the radius. I saw in one case a reflex contraction produced in the deltoid when the capitulum ulnæ was lightly tapped, and one in the triceps brachii when a spot near the c. ulnæ was tapped. In all these cases the experiments were carefully repeated, to show that the reflex action did not originate in the skin, and that no mechanical shock, transferred to the distant muscle, could have been the cause. In hemiplegic patients, when the patellar tendon of one side is tapped, we often see twitching of the adductors of the other

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side. In a case of compression of the lumbar cord the tendo-patellæ reflex action was absent; when the motility returned, the reflex action also appeared—a proof that the integrity of the conduction to the central organ is requisite. In tabes we often find the tendo-patellæ reflex action wholly absent, while the mechanical excitability of the quadriceps is retained or even exaggerated.

But the question has since been attacked experimentally, and, as it seems to me, has been decided beyond a doubt. F. Schultze and P. Fuerbringer[1] have made a series of experiments, all favorable to the reflex theory. It appeared, first, that in rabbits and dogs the phenomenon of the tendo-patellæ is a quite constant occurrence, and has remained unknown to physiology only because it has never been looked into; the reflex act can be produced with especial ease and distinctness from the exposed tendon. The experiments were associated in some instances with section of the spinal cord, in others not; they were modified in the greatest variety of ways by section of the nerves and muscles, poisoning with curare, etc., and their uniform testimony was to the effect *that the muscular phenomena in question cannot be due to a direct action through the tendon, but depend on a reflex operation*, the mechanism for which, in the case of the lower extremity, is situated in the lower segments of the spinal cord; and lastly, *that a reflex act originating in the skin cannot possibly be the cause.*

The last statement leads by anticipation to the observation of Joffroy[2] that these phenomena, at least in pathological cases, are essentially due to irritation of the skin, while the irritation of the tendon plays only a subordinate part. Joffroy brings many instances in which irritation of the skin produced the phenomenon of reflex clonus of the muscles of the calf, to be described below. I have repeatedly seen this. I have, however, convinced myself that this also is due simply to a secondary irritation of the tendon. In such cases, every cutaneous irritation of the foot produces a reflex dorsal flexion of the member, and this suffices to originate a reflex clonus, exactly as passive dorsal flexion would produce it. Whether or not there are cases in which the spasmodic tremor can be originated directly from the skin, I must for the present leave undecided.

It remains to be said, that O. Berger is decidedly in favor of the reflex theory, upon the ground of such facts as I have mentioned.[3]

We may therefore with propriety introduce the name of "tendinous reflexion" for this phenomenon.

Our view is decidedly supported by C. Sachs' very recent demonstration of nerves in the tendons, which can hardly have any other than a sensitive function.[4]

It is also known, and has lately been confirmed by Freusberg's data, that numerous reflex actions may originate from the viscera, as the bladder, rectum, anus, the intestines (dependent on their degree of fulness), etc. Freusberg has, finally, tried to show the probability that reflex actions may also be originated from sensitive nerves in muscles, by twitching and stretching them. All these matters find their parallel in human pathology.

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[1] Centralbl. f. d. med. Wiss. 1875. No. LIV.

[2] De la trépidation épileptoïde du membre infér., etc. Gaz. méd. de Paris. 1875. No. 33 et 35.

[3] Schles. Gesellsch. f. vat. Cult. Medic. Sect. Sitzg. v. 23. Juli, 1875.

[4] Die Nerven der Sehnen. *Reichert* und *Du Bois*' Archiv. 1875. p. 402.

We have hitherto spoken almost exclusively of reflex actions which affect the voluntary striped muscle. But it is easy to show that reflex actions may extend to all the centrifugal phenomena, and that such actions play a most prominent part in the occurrence of many phenomena of movement; we would call to mind the reflex processes which are so essential to the discharge of fæces and urine, to the movements of the stomach and intestine, to erection and ejaculation, to the movements of the uterus; the reflex production of the secretion; and lastly, the important reflex processes which occur in the blood-vessels, and are carried out through the vaso-motor paths.

Inhibition of Reflex Action.

The experiments upon reflex action have also shown that the irritation of certain parts may give rise to an inhibition or suppression of spinal reflex acts. And it appears that this effect may be produced as follows:

First, a powerful arresting influence proceeds from the *brain* (Setchenow). Daily experience shows that we can suppress many reflex acts by the aid of the will; but this relates only to such acts as stand under the general control of the will, and can be performed by a voluntary effort. Experiments show that separation of the brain from the cord considerably increases the spinal reflex actions; such a separation is in fact always made when the reflex processes have to be studied. It is further shown that irritation of certain parts of the brain (in frogs, the so-called optic lobes) lessens the spinal reflex acts, and retards them, or entirely puts an end to them. The paths of conduction for these inhibitory influences from the brain are thought to lie in the white anterior columns.

Inhibition of reflex acts can also be produced from the *periphery.* Numerous physiological experiments within the last few years have shown that the spinal reflexions can be arrested and completely suppressed by *irritation of sensitive nerves* (Lewisson, Setchenow, Nothnagel, Goltz, Freusberg). A great variety of sensitive paths may be used by this function; the inhibition takes place with most certainty from the skin, whether by powerful irritation of a limited spot, or by slight irritation of large surfaces; it may also be originated by irritation of the sensory nerve-trunks, of the sensitive muscular nerves, or of the viscera (*e. g.*, by distention of the intestines and stomach). The paths used in the production of these processes of inhibition lie, without doubt, in the posterior roots.

We have, however, nothing but conjecture to aid us in forming an idea as to how these acts of inhibition come to pass in the cord itself. We know that the reflex acts are arrested when the sensory cells of the reflex arc receive impressions simultaneously from other sensory districts—central or peripheral. "The susceptibility of certain centres to impressions which give rise to the reflex act is lessened, when these centres receive impressions from other nerves at the same time" (Goltz). It is quite obvious that this statement is not an adequate explanation. Perhaps there exists a special inhibitory apparatus in the cord.

Centres and Paths for the Innervation of the Viscera.

The innervation of the heart, apart from the centres situated within itself, is dependent on certain centres in the medulla oblongata. As regards the part taken by the cord in the extremely complicated innervation of the heart, there still exists a difference of opinion. It is thought that the exciting centre for the movements of the heart is situated, partly or wholly, in the upper cervical region, and that the paths which lead from it run downward for some distance in the cord, after which they reach the sympathetic by various routes, thence passing to the heart. Irritation of the paths in the cervical cord is said to accelerate the activity of the heart.

As regards those roots of the accessorius, which originate low down in the cervical cord, it is not yet clear how important they may be in the innervation of the heart. But the cord possesses a powerful influence on the movements of the heart, through the vaso-motor innervation ; it is well known that irritation or paralysis of the vaso-motor nerves exercises a great influence upon the rate and the force of the heart's action.

The activity of the *organs of respiration* is also dependent on the respiratory centres in the medulla oblongata. Recent investigations by P. Rokitansky[1] seem to show that there exist respiratory centres, analogous to the vaso-motor, in the upper part of the cord, whose function becomes more distinct after the cord is severed from the medulla oblongata.

The paths which conduct the excitation from the centres of respiration to the muscles of respiration are said to be all contained in the lateral columns of the cervical cord and upper dorsal cord. This view has lately been maintained by Schiff against the attacks of Brown-Séquard and others.

The cord seems to have a great influence upon the movements of the *digestive tract.* All these movements (swallowing, peristole of the stomach and intestine) are reflex in their nature, and are probably produced by centres situated in the cord. On the other hand, the cord has also a reflex inhibitory power over these movements. Thus Goltz[2] has demonstrated an inhibitory influence exercised by the cord upon the movements of the œsophagus and the stomach, and states further, that destruction of the cord produces extensive and active peristaltic action of the bowel, and causes diarrhœa. It is much to be wished that we possessed more accurate investigations into these relationships, and into the seat of the centres and paths.

The *evacuation of the rectum* is produced by a more complicated mechanism, as follows : The contents of the intestine enter the rectum, producing a reflex peristaltic action of the latter; the centre for this reflex act is situated in the lumbar cord. The pressure of the contents against the place of exit probably at first excites the tonicity of the sphincters by reflex action, and interferes with evacuation. At the same time the sensitive nerves give notice to the consciousness of the approach of an evacuation, and the influence of the will can be invoked to strengthen the contraction of the sphincter and prevent the occurrence of discharge for a while. If the reflex contraction becomes stronger, or if the sphincter is voluntarily relaxed, the discharge takes place. It is aided by the action of the abdominal compression (straining), which is either voluntary, or, in case of severe irritation of the mucous membrane of the rectum (tenesmus), is directly reflex. The passage of the masses of fæces through the anus provokes those rhythmic reflex contractions which Goltz[1] has described, the reflex centres for which must also be sought in the lumbar cord. These contractions close up the rectum.

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[1] Untersuch. üb. d. Athemnervencentra. Wien. med. Jahrb. 1874. I. S. 30.

[2] Pflüger's Archiv. VI. 1872.

The centres for all these processes lie in the lumbar cord; the paths from the centres to the rectum lie in the sacral nerves and the sympathetic plexuses. And as the sensory and motor paths which ascend from the rectum through the cord to the brain (the position of which in the transverse section is not yet exactly known), are also concerned in the process of fæcal evacuation, it is easy to see how many points there are whence pathological disturbances in the process of evacuation may originate.

Quite analogous conditions are found in the *evacuation of urine*, the disturbances of which are so extremely common in diseases of the cord. According to Goltz' new and admirable investigations,[2] the normal process is as follows: the increasing fullness of the bladder produces an increasing irritation of the walls; by this sensory irritation a reflex contraction of the detrusor is occasioned through the agency of a centre located in the lumbar medulla; at the same time the impulse to urinate reaches the consciousness, when the evacuation may be prevented by voluntary contraction of the sphincter vesicæ, or of the urethral muscles which act as sphincters, until the vesical muscles become fatigued and the impulse to urinate diminishes. The tonus of the sphincter may also, perhaps, be increased reflexly by the entrance of the first drops of urine into the urethra. After some time fresh and more powerful contractions of the detrusor occur, until at last the sphincter is overpowered, or is voluntarily relaxed, when the evacuation takes place; it can be hastened by the action of abdominal straining, either voluntary, or, if the impulse to urinate is very strong, of a reflex sort; and is concluded by a few rhythmic contractions of the urethral muscles.

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[1] Pflüger's Archiv. VIII. 1873.

[2] Ueber die Functionen des Lendenmarks des Hundes. Ibid. VIII. p. 474.

The act of evacuating the bladder is, therefore, purely reflex in its nature; the centre for its accomplishment lies in the lumbar medulla.

After section of the dorsal medulla the evacuation takes place in a perfectly regular way, whenever the bladder has reached its normal point of fullness, or the wall of the bladder is irritated in some other way. The complete paralysis and cessation of evacuation which appears to occur during the first days after the operation, depends on the concussion (Erschütterung) of the lumbar medulla and paralysis of its centres which the operation brings about. These centres usually recover in a short time, and resume their functions.

The evacuation is excited by sensory irritations, the most active of which is irritation of the wall of the bladder itself by distention and pressure from its contents; but irritation of the anal region may also provoke the discharge. The sensory and motor nerves of the bladder, which form the paths for this reflex process, leave the lumbar medulla along with the roots of the sacral nerves (probably the third, fourth, and fifth), and pass with them, either directly or through the sympathetic plexuses, to the mucous membrane and the muscles of the bladder and the urethra.

There are, however, other motor and sensory paths, which lead upwards in the spinal cord from the bladder to the brain. Budge has succeeded in producing contractions of the bladder by stimulating the cord as high up as the pedunculi cerebri; the paths are supposed to lie in the anterior columns of the cord. It requires no proof to show that the routes for the voluntary excitation of the sphincter and the urethral muscles also run through the cord to the brain.

In spite of this, the will seems to exercise no direct influence upon the contraction of the detrusor. The voluntary discharge which we can produce without the presence of the impulse to urinate, is probably brought about by relaxing the sphincter, and bringing a powerful abdominal pressure to bear upon the walls of the bladder, which gives rise to a reflex contraction of the detrusor (Goltz). But that by an involuntary reflex action originating in the brain the reflex mechanism of the lumbar medulla may be brought into activity, and an evacuation produced, is shown by the cases in which psychical impressions are followed by sudden discharge of urine; also by the fact that certain ideas are capable of calling out the desire to urinate, or of increasing it considerably. The paths in the spinal cord which serve for the propagation of this class of impressions may be excited by irritating the cord itself, and contractions of the bladder produced.

It is a matter of daily observation, and hardly needs to be mentioned, that inhibitory processes exist in the case of the mechanism of urination, as of all other reflex actions.

The correctness of the above statements can easily be shown by careful observation of one's own person, and is fully confirmed by pathological facts. We should, however, be careful to observe that disturbances in the function may originate, not only in the centre in the lumbar cord, but also in the sensory and motor paths which unite the bladder with this centre on the one hand, and with the brain on the other. The relations may certainly be very complicated.

Very similar conditions are met with in the processes of *erection* and *ejaculation*, which also depend mostly upon the spinal cord, and which have received fresh light from the observations of Goltz (l. c.).

The erection of the penis is brought about, according to Eckhard's investigations,[1] by direct irritation of the so-called nervi erigentes, which originate in the sacral plexus and are distributed in the corpora cavernosa. The process is now universally regarded as an act of inhibition, exercised by the nervi erigentes upon the ganglionic apparatus of the vessels of the penis (Lovén); this causes a relaxation of the vascular tone, and a powerful influx of blood into the corpora cavernosa, which produces the erection.

This irritation of the nervi erigentes occurs in the reflex way also; the centre for this reflex act lies in the lumbar cord (Goltz), for it is very easy to produce reflex erections in dogs after section of the dorsal medulla.

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[1] Beitr. z. Anatomie und Physiologie. Giessen. Bd. III. IV. und VII.

This reflex act is produced with most certainty by irritation (slight friction) of the skin of the penis and glans, or of the skin of the lower abdomen and perineum; by irritating the bladder or rectum, by introducing the catheter, and probably also by irritation of the testes, by over-fullness of the seminal vesicles, etc.

The reflex act can be arrested or suppressed, either by powerful peripheral irritations or by cerebral influence. Complete destruction of the lumbar cord renders it impossible.

The brain also possesses a certain influence upon the occurrence of erections; but this is not a direct influence of the will, for erections cannot be thus produced. But erections can be produced by loose thoughts, by stimulation of the fancy, by looking at things which excite sexual appetite. This appetite is known to be located in the brain; from the cerebral centre, the mechanical reflex centre in the lumbar cord may receive the exciting impulse. The paths which convey this excitation from the brain to the lumbar cord must lie in the spinal cord. In fact, Eckhard has succeeded in producing erections by irritation of the spinal cord, as high as the pons and pedunculi. The same is the case in many diseases of the spinal cord. The portion of the transverse section of the cord which contains these paths is not yet known.

Quite the same processes are passed through in *ejaculation;* for this also is a simple reflex act having its centre in the lumbar cord. It seems, however, to require for its production a somewhat longer and more powerful irritation. The nerve-paths probably lie chiefly in the sacral plexus.

The spinal cord has also an influence upon the *uterine contractions.* The motor nerves of the uterus lie in the spinal cord, and may be followed, by irritating them, up into the medulla oblongata (W. Schlesinger[1]). Uterine movements may also be provoked reflexly from the sciatic nerve. The centre for these movements does not lie exclusively in the medulla oblongata, as was formerly thought, but such centres may be demonstrated in the whole length of the cord (Schlesinger). The chief centre for the production of labor-pains lies in the lumbar cord, according to Goltz.[1] After division of the dorsal medulla the reflex acts proper to copulation, and those of labor and birth take place in a normal manner. The processes of ovulation, of development of the pregnant uterus and the lacteal glands, the development of the impulses which are associated with reproduction, suffer no visible impairment from this operation.

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[1] Ueb. d. Centra der Gefäss- u. Uterusnerven. Wien. med. Jahrb. 1874. I. p. 1.

Nasse has observed in the human subject, after crushing of the cervical medulla, a normal performance of the act of parturition.

The *innervation of the iris* is also in part dependent on the spinal cord. The motor-paths for the dilatator pupillæ lie in the cervical and upper dorsal cord. Irritation of this region produces dilatation of the pupil; it has therefore received from Budge the name of centrum cilio-spinale. According to Salkowski (Dissert. Königsb. 1867), this centre lies much higher up, namely, in the medulla oblongata. The motor-fibres which pass from it run downwards in the cord without decussating, pass out with the anterior roots of the lower cervical and upper dorsal nerves, go thence into the cervical sympathetic, and then to the eye. Section of these fibres causes contraction of the pupil. The same route is taken by the vaso-motor paths for the head and external ear.

The influence of the cord upon the various *processes of secretion* in the body has been very little studied. It is probable that there exists such an influence upon the secretion of sweat and saliva, and doubtless also upon the production of semen and ovulation, the secretion of the digestive fluids, etc. But at present we are not in possession of exact knowledge in respect to these points.

The only positive facts of this sort that we possess are those furnished by Eckhard[2] in regard to the secretion of urine, who says that section of the cervical cord produces a complete and permanent arrest of this secretion. Eckhard infers from his experiments the existence of a centre for exciting the secretion of urine, situated at the level of the rhomboid fossa; the excitor paths which pass downwards from this centre leave the cord by the upper dorsal nerves; there are also inhibitory paths for the secretion of the urine, located in the splanchnic nerve. The qualitative changes in the urine, which are so common in affections of the spinal cord, are probably due in great part to stagnation of the urine in the paralyzed bladder, and to the decomposing influences of secondary disease of the bladder.

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[1] Pflüger's Archiv. Bd. IX. p. 552.

[2] Untersuchungen über Hydrurie. Beitr. zur Anatomie und Physiologie. Bd. V. p. 147. 1870.

The theory of *muscular tonicity* needs only a very short mention. It has called forth a great deal of investigation, but can hardly be applied in pathology. We understand by the term muscular tonus, a constant mild innervation of the striped muscles by a stimulant influence proceeding from the spinal cord. Later investigations have shown that this is probably in substance nothing more than a weak reflex excitement, which originates in sensory stimulation of the skin, muscles, joints, and other parts, and is chiefly produced by the action of changing the position of the members of the body.

In connection with this point stands the much discussed question of the *influence of the posterior roots on the excitability of the anterior.* While some physiologists (Harless, Cyon, Steinmann, etc.) state with perfect confidence that the excitability of the anterior roots is depressed after section of the posterior, other observers (v. Bezold, Uspensky, G. Heidenhain, and others) have denied the statement with equal confidence. But even if the fact were proved, it does not possess the great importance claimed for it in pathology.

A *tonus of the vascular muscles* seems proved. The vasomotor nerves are its conducting paths; they are kept in constant slight excitement by the centres demonstrated in the medulla oblongata and the spinal cord, and, after these are removed, by peripheral ganglionic apparatuses, which also have the power of keeping up the tone of the vessels.

The *tone of the sphincters* of the bladder and rectum is certainly of a reflex character, and principally depends on the lumbar cord.

The application of physiological data to pathology is certainly of the greatest importance; it is the source of the only light that has been cast on a great number of pathological processes and their connection. It must be admitted, however, that physiology is far from explaining everything, and that many points can only be made clear by pathological observations and experiments.

Physiological experiments very often produce a pathological state (section, compression, irritation, etc.); similar states are occasionally produced in man by a great variety of agencies, including disease; and these are the cases in which a direct application of physiological laws to pathology will prove most fruitful of results.

But the states produced in physiological experiments are far from being so various and general, and are seldom so exactly localized, as are pathological conditions. From the nature of the case they must be very limited in extent; the usual lesions consist of severance of continuity, very small, and with hardly any extension in the direction of the long axis of the cord. Simple sections have hitherto formed almost our entire foundation for the experimental pathology of the cord; and it is time that the method of Nothnagel and Fournié, applied with such success to the brain, were extended to the spinal cord.

A brief consideration shows that the experiments of physiology and pathology can imitate neither those anomalies which are widely distributed along the length of the cord, yet localized in particular spots; nor the slowly progressive conditions of irritation and paralysis; nor the moderate and gradually increasing and varying degrees of pressure; nor the various finer disturbances of nutrition in fibres and cells.

In particular, it must be noted that possibly, even probably, the properties of irritability inherent in the cord may be essentially changed by pathological processes, so that, for instance, the kinesodic substance becomes motor, the æsthesodic sensitive. Thus conclusions, drawn from the healthy cord, cannot be transferred to the same organ in a morbid state, without certain reserves.

These reasons may justify the objections which have been made to a direct transference of physiological principles (in many respects poorly founded) to the pathology of the cord. Nevertheless, we think it not unsuitable to collect in this place some of the principles deduced from physiological and pathological experience, so far as they seem applicable to practical needs, as a sort of clew to the interpretation and recognition of complicated pathological processes.[1]

1. Section or limited affection of the posterior columns destroys the sense of touch in parts situated behind the point of injury, but leaves the sense of pain.

2. Disturbance of the conductive power of the gray substance for a limited longitudinal extent suspends the sense of pain, but leaves that of touch (analgesia).

3. Disease or destruction of the entering posterior root-fibres (or of the network of fibres directly formed by them) must impair the sense of touch equally with that of pain and the other classes of sensation.

4. Injury or disease of the posterior columns at the level of the lumbar cord leads to a diminution of the sense of touch at the anus, perineum, etc., while the sensibility and motility of the lower extremities remain unimpaired; the same lesions in the lateral columns of the lumbar cord have the same effect upon the lower extremities as those of the posterior columns in the dorsal and cervical medulla.

5. When the gray substance is partially destroyed in the transverse direction, and the posterior columns are also affected, the conduction of sensory impressions is retarded, in a degree proportional to the smallness of the transverse piece of gray matter that remains. But if the conductive power of the posterior columns is retained, this retardation appears to extend only to the sensation of pain, while the conduction of the sensation of touch takes places with normal rapidity.

6. Destruction of the entire extent of the posterior columns (inclusive of the sensitive root-fibres passing through them) must be followed by anæsthesia of a corresponding extent.

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[1] Compare the "Corollarien für die Pathologie," given by *Schiff* (Physiologie, p. 292), and *Brown-Séquard's* statements in his Course of Lectures on the Physiology and Pathology of the Central Nervous System. 1860.

7. Limited destruction of the entire transverse extent of the posterior columns and of the gray substance is followed by complete anæsthesia of the portions of the body lying posteriorly, and weakness of motion or partial paralysis.

8. An *irritation* affecting a limited longitudinal extent of the posterior columns (inflammation, hyperæmia, etc.), produces a spontaneous pain in only those roots which traverse the diseased spot (girdle-pain); subjective sensations of touch (formication, prickling, numbness, sensation of heat and cold) and some degree of hyperæsthesia occur in the parts situated posteriorly.

9. A *lesion producing paralysis*, affecting the posterior columns in the same way, gives rise to a girdle of complete insensibility, corresponding to the district supplied by the paralyzed nerve-roots; below this girdle the so-called sensations of touch are absent, or greatly impaired; the sensation of pain is retained, but is badly localized.

10. If an affection which at first irritates, and afterwards paralyzes, progresses upwards, the painful girdle travels upwards, and leaves behind it a girdle of anæsthesia which gradually increases in width; in the parts situated behind this the sense of touch is gone, but subjective impressions of touch (formication, numbness, etc.) may be present.

11. When the power of movement is unimpaired, and a girdle of pain without aberration of the sense of touch is present, then only the nerve-roots, within or without the cord, are affected.

12. In diseases of the posterior columns and the gray substance, the parts behind the diseased portion may experience only changes in the sense of touch, without any excentric pains. (?) If the latter occur, they point to an implication of those nerve-roots which are situated further back.

13. Disorganization of an anterior and a lateral column and of the greatest part of the gray substance produces paralysis of the same side.

14. Destruction of the anterior (and lateral) columns in their entire transverse section (inclusive of the motor nerve-roots passing through them) is followed by a paralysis of corresponding extent.

15. Limited destruction of the entire transverse section of the

anterior (and lateral) columns and of the gray substance is followed by complete paralysis, also analgesia, but retention of the sense of touch.

16. Disease of the antero-lateral columns and the kinesodic substance alone produces paralysis without lesion of sensibility.

17. Disease of the motor ganglia, into which the motor roots first enter, produces paralysis in the region of the related nerves, without disturbance of sensibility, but with trophic disturbances.

18. Affections of the antero-lateral columns and the corresponding gray substance produce contracture or convulsions *only* in the muscles immediately dependent on the diseased spot and its motor roots; but contractures of muscles supplied by the roots given off behind the affected spot are not produced (?).

19. Slight pressure on the cord may bring on paralysis of the extensors and secondary contractures in flexion, but this is never severe.

20. Contractures and convulsions of the lower extremities also occur in affections of the segments of the cord above the lumbar region; they are then a consequence of an implication of the posterior columns, and arise reflexly. In the same way, in diseases of the posterior columns, spasmodic symptoms occur in the parts situated nearer the head.

21. Disorganization of the entire gray substance to a considerable distance must be followed by anæsthesia and paralysis in the posterior part of the body; if the lesion is limited to one place, the sensory and motor paralysis may be partial.

22. If the movements of respiration are entirely intact in an affection of the cervical cord which paralyzes the extremities and trunk, then the lateral columns are not involved.

23. Conditions of irritation in the cervical medulla will produce dilatation of the pupil; paralytic conditions, contraction.

24. Unilateral lesion of the cord is followed by almost total paralysis and increased sensory excitability on the injured side, with very slight disturbances of motion and loss of sensibility on the opposite side.

25. Complete compression or division of the spinal cord exaggerates the reflex acts in the region lying posteriorly to the lesion.

26. In limited destruction of the dorsal medulla, the reflex acts which are performed through the lumbar cord (evacuation of urine and fæces, vascular tonus, etc.) go on with very little alteration ; only they can no longer be modified by the will.

27. The nutrition of peripheral parts (muscles, nerves, bones, joints, skin, etc.) remains intact in the various diseases of the spinal cord, in proportion as the gray substance remains normal.

The doctrine of the *functional reparation* (Ausgleichung) *of partial lesions of the cord*, stated by Schiff, is of great importance in pathology; the fact, namely, that while these lesions are not anatomically made good, an apparently complete restoration of function follows. Schiff[1] affirms that in injury of almost any part of the cord, the consequent functional disturbances may be compensated for by an intact portion of the cord assuming the function of the injured portion; the lesion of the posterior columns *alone* leads to a *permanent* loss of the sense of touch, which cannot be repaired.

The chief element in this functional restoration is of course a vicarious assumption of the processes of conduction by intact portions of the cord. This fact is not of itself strange, for we can observe in the fine network of nerves what seems to be the anatomical provision for such exigencies.

It is not yet settled how far such a vicarious substitution may go in human pathology; but it is clear how wide must be its significance in relation to the prognosis and cure of partial lesions of the cord.

It will be proper to introduce here a few remarks on the *anatomical restoration* of partial lesions of the cord. Daily experience shows that this happens very often, and may be quite considerable in amount; it is not rare for apparently very severe injuries of the cord to recover. But the exact histological processes are not known; it is not yet well ascertained how a chronic inflammation, or the various degenerative processes, scleroses, softening, hemorrhage, etc., are repaired, nor to what extent this takes place.

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[1] Centralbl. f. d. med. Wiss. 1872. No. 49.

Experimentally, this question has been very little examined, although the physiologists have had material enough. A few positive facts were ascertained by Flourens, Brown-Séquard, H. Mueller; but in recent years Masius and Vanlair[1] have been the first to institute thorough experiments upon frogs, showing after a lapse of at least six months a great progress in restoration of excised segments of the spinal cord. Motility and sensibility were restored, and nerve-cells and fibres were found in the cicatrix. In the higher order of animals, especially the mammalia, the restoration seems to be less easy and perfect. The same result appears from the latest experiments of Eichhorst and Naunyn[2] upon very young dogs. After section or crushing of the lower dorsal cord, the first occurrence is a complete degeneration and fluidification of the parts directly attacked; afterwards an intermediate substance, composed of a tissue like neuroglia, rich in cells, is developed, which incloses a central cavity. Subsequently the double-contoured nerve-fibres are regenerated, and a limited number are seen to traverse the intermediate substance. Regeneration of the ganglion cells was never observed. Corresponding with these conditions, a partial restoration of function appears after many weeks (eight or ten, at least); voluntary, but incomplete and "atactic," movements are the first to appear, and sensibility returns later. But the animals die subsequently, probably in consequence of a secondary hydromyelus.

On the other hand, Goltz and Freusberg have never seen a regeneration and restoration of function in their numerous experiments upon dogs, although some were kept living for an extremely long time. For this reason Freusberg cannot avoid expressing a suspicion of the correctness of the results, as regards restoration of function, obtained by Naunyn and Eichhorst.[3]

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[1] Centralbl. f. d. med. Wiss. 1869. No. 39; and Arch. de Physiol. norm. et path. IV. p. 268.

[2] Arch. f. experim. Pathol. und Pharmacol. Bd. II. p. 225. 1874.

[3] Pflueger's Arch. Bd. IX. p. 390.

It certainly follows from these experiments that in the higher animals, and probably in man also, the regeneration of the spinal cord, when once completely destroyed, will always remain very imperfect, even if it takes place to any extent.

III. General Pathology of the Spinal Cord.

In this section we design to give a short account of the facts and observations, but only as far as they seem to possess a present interest and importance in relation to practice. We shall lay the chief weight upon general symptomatology and therapeutics, and shall take leave to pass over the general pathological anatomy, which at present is not in a condition to be made useful to the practitioner. General etiology and diagnosis will be but briefly touched, in view of their present condition.

A. General Symptoms of Diseases of the Spinal Cord.

This involves a systematic enumeration of the several disturbances which occur in diseases of the cord, partly in order to explain their significance and names, partly to reduce them to their immediate causes and develop their pathology, partly to point out somewhat in anticipation the more usual groupings of symptoms. We shall thus be enabled to save many repetitions and details in the special part.

1. *Disturbances of Sensibility.*

These are very frequent, and their form and grouping is often very characteristic. They are very important in diagnosticating and estimating diseased processes in the cord; for these reasons they must be stated with great fulness.

In examining sensory disturbances, the several qualities of sensation must be strictly distinguished. Let the sensations of touch, temperature, and tickling, of pressure, of space, and of pain be tested. As regards the best methods of doing this, compare Vol. XI. of this Cyclopædia, p. 212 [American ed.]. The qualities of perception which are classed together under the name of muscular sensation, or sense, should also be tested. Besides the methods given in the same volume, p. 234, there is a good method given by Leyden[1] for the exact testing of the sensation of passive motions. No extensive apparatus is required for this; the same exact results are obtained when the leg is suspended in a broad cloth, and by means of the cloth is made to move in various directions, upwards, downwards, outwards, or inwards—its position being either extended (for testing the hip-joint) or semi-flexed (for the knee-joint). The patient is required to state the extent and direction of the movements. In testing the passive movements of the ankle-joint, the front part of the foot is grasped carefully with the hand, and is moved passively. This method is quite sufficient, as the cutaneous sensibility of the patient is usually impaired.

a. Diminution of Sensory Action—Anæsthesia.

All the sensations which originate in the skin, the muscles, and other more deeply-seated parts may be diminished in diseases of the spinal cord; and the diminution may increase, even to the extent of total loss of sensation. They may be extinguished all at once, or some may be lost and the rest retained.

The disturbance of sensibility usually appears first in the lower extremities, ascending by degrees until it reaches the upper extremities. But often the latter are the first attacked, and the anæsthesia extends downwards.

Anæsthesia is very commonly attended by various subjective sensations, as the furry sensation (Pelzigsein) or numbness, uncertainty in feeling the ground, sensation of walking on cotton wadding, or on a bladder full of water, etc.

In general, the occurrence of anæsthesia permits us to infer an implication of the posterior half of the cord.

A widely diffused, total paralysis of sensation (total = affecting all the qualities) occurs only when the entire transverse section of the posterior columns and the gray substance is destroyed; that is, chiefly in affections which are diffused over the entire section, extending to a variable distance in the longitudinal direction. It also accompanies a complete severance, crushing, or compression of the cord at any point; in which case the anæsthesia occupies all portions of the body which derive their nerve-supply from behind the point of lesion.

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[1] Ueber Muskelsinn und Ataxie. Virch. Arch. Bd. 47. 1869.

Total paralysis of sensation, of a more limited extent, may occur in various ways, viz.:

As *unilateral anæsthesia*, localized in one leg, or a leg and the corresponding half of the trunk, or finally, in these parts and the corresponding arm. This occurs in traumatic or spontaneous unilateral lesion of the cord, and the loss of sensation takes place on the side of the body opposite to the spinal lesion, owing to the decussation of the sensory paths in the cord. The muscular sense, however, usually remains unimpaired, because the fibres for it cross at a higher point.

As *anæsthesia in the form of a girdle*—a zone of varying width, deprived of sensation, which surrounds at various levels the pelvis, the abdomen, the thorax, or even the region of the shoulder or neck on one or both sides. It is due to a local disease of the posterior roots within or without the medulla, which extends over but a limited length of the cord ; or to a circumscribed affection of the gray posterior cornua, embracing the network of nerve-fibre which is formed by the entering root-fibres, and the paths which traverse the gray substance before re-entering the posterior columns.

Finally, as *circumscript anæsthesia*, limited to certain extremities or parts of extremities, or to the district supplied by certain nerve-trunks. The most frequent cause of this lies probably in affections of single bundles of roots ; it may be produced by local affections, limited to portions of the transverse section of the spinal cord, and affecting only certain of the longitudinal fibres, but this lesion would be more likely to produce a partial paralysis of sensation. It is probable that the sensitive paths for the upper and lower extremities, the anterior and posterior surfaces of the body, etc., have a distinct arrangement in the spinal cord ; and it may easily be imagined how many forms may be taken by such circumscript anæsthesia, according to the horizontal or vertical distribution of the morbid change.

Partial paralyses of sensation, however (limited to certain of the qualities of sensation), also occur, and nowhere more frequently than in diseases of the cord ; the history of tabes dorsalis gives the most numerous examples of this. They occur in all possible combinations, as indicated in Vol. XI., p. 201. The form most likely to attract notice, and perhaps the most frequent, is *analgesia;* but, as remarked, the greatest variety of cases of partial anæsthesia occur. Each sort of sensation may alone be lost or weakened; and, on the other hand, several may suffer the same change, while only a single one exists in partial or complete integrity.

In view of these facts, we can hardly avoid the conclusion that the various sensations traverse distinct paths of conduction in the cord, and that, according to the local distribution of the morbid change in transverse section, sometimes one and sometimes another path is specially affected. Nothing exact is known, however, in respect to this. It seems probable that the sense of pain is conducted by the gray substance only, and impressions of touch by the posterior columns only (Schiff). In opposition to this view Brown-Séquard asserts that all sensations are chiefly conveyed through the gray substance, and he even names distinct regions of the latter, which are supposed to contain the respective groups of fibres. The result, therefore, will differ in each case, according to the manner in which the disease is distributed over the various parts of the transverse section of the cord.

But little of practical value can be drawn from these scanty and uncertain facts. If disturbances of sensation are present, the physician will have to put to himself the questions—whether there is an affection of the posterior roots within or without the cord, or whether there is an impediment to conduction within the gray substance, or whether certain sensory paths have been injured at a higher point, after leaving the gray substance. The data which are given here and in the physiological introduction will show the points which aid us in making these distinctions; but they will also show how few and unsatisfactory these points are.

What is true of cutaneous sensation may be likewise affirmed of the so-called *muscular sensation;* both the muscular sense and that which is called the muscular sensibility[1] may be reduced or suspended in spinal diseases. The patients lose the sensation of pain in the muscles, produced by various external agencies; they lose the sense of fatigue; they are not certain of the position of their limbs in the dark, or when their eyes are closed; they have lost the feeling of passive movement of the limbs, their ability to retain their equilibrium is diminished, etc.

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[1] Compare Volume XI. p. 233.

Regarding the position of the routes which transmit these impressions in the spinal cord, we know very little. According to Brown-Séquard, at least a part of them remain on the same side of the cord, and do not decussate till they reach the medulla oblongata. The application to pathology is evident.

A symptom which is not very rare under physiological conditions is *the retardation of the conduction of sensory impressions*. This remarkable fact was first mentioned by Cruveilhier[1] without accounts of special cases; since then, although often observed, it has never been closely studied until very lately, and even now the investigation is far from complete.

This retardation is very noticeable and measurable. While in ordinary circumstances sensation follows directly upon the application of the stimulus, in cases like this it is separated from the latter by a noticeable interval of time, which is frequently a fractional part of a second, but not rarely amounts to one or several seconds; cases have even been observed where the sensation came from fifteen to twenty seconds later than the stimulation (Cruveilhier), thirty seconds (Topinard), and even several minutes. In such cases the phenomenon is of course very easily observed; in less marked cases the existence and the degree of retardation can be determined by exact measurements, such as have been made by Leyden and Goltz.[2] The more powerful the stimulus, the shorter the interval.

It has been repeatedly observed, and very recently established with exactness, that this retardation relates only to a few of the qualities of sensation, chiefly to that of pain. E. Remak[3] has published a case in which pricking with a needle provoked in every instance an immediate sensation of touch, which was followed in three seconds by the retarded perception of pain. In such cases every powerful impression gives rise to a double sensation, first, one of touch, perceived with normal quickness, and then a retarded sensation of pain. The case published by Naunyn in the same journal[1] seems on some accounts to belong here; it included retardation of the sensation of pain, associated with hyperæsthesia, while the sensation of touch remained normal. Vulpian[2] observed a similar state in a case of tabes ending in apoplexy; the prick of a pin was rapidly followed by a slight reflex action, and two or three seconds later by a very full and continued movement of defence. I have under my observation at present a tabetic patient, in whom I have been able to demonstrate this double sensation, not only as regards needle-pricks and pinching, but also under the painful faradic current.

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[1] Anatom. pathol. Livrais. XXXVIII. p. 9.

[2] *Leyden*, Klinik der Rückenmarkskrankheiten. I. p. 146.

[3] Arch. f. Psych. u. Nervenkr. Bd. IV. p. 763. 1874.

E. Remak has treated very thoroughly of the question, whether this retardation of conduction is always limited to the sense of pain, and never affects that of touch. The observations hitherto made seem almost to show that such is the case; but the question needs further careful study, and it is hard to give a reason why the sense of touch should not in some cases be affected. The cases commonly tested are those of tabes dorsalis, in which the sense of touch is more or less weakened, while that of pain is retained; if both are retained, the double sensation may occur. Topinard also states that the retardation affects chiefly the senses of pain and of temperature.[3]

It has been made known through the physiological researches of Schiff, that a transverse narrowing of the gray substance (whether the posterior columns are cut or no) produces a corresponding retardation of the conduction of sensation, which is marked in inverse proportion to the amount of gray matter remaining.[4] Schiff has even been enabled by his experience to predict the existence of double sensation in man, which has lately been confirmed by the beautiful observation of E. Remak; he looks for the appearance of this symptom in every case where the transverse dimensions of the gray substance have been diminished by pathological processes, while the posterior columns have remained intact.[1]

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[1] Archiv f. Psych. u. Nrevenkr. Bd. IV. p. 760.

[2] Arch. de Physiol. norm. et path. I. p. 405.

[3] *Hertzberg* (Beitr. zur Kenntniss der Sensibilitätsstörungen bei Tabes. Diss. Jena. 1875) has lately demonstrated, in some very carefully examined cases, that the most frequent occurrence is that of retardation of the sense of pain *alone*, but that the sensations of touch and temperature also are not rarely retarded, though to a less degree.

[4] See *Schiff's* Physiologie. p. 245.

It may be assumed, on the strength of these facts, that wherever retarded sensation exists, there is an *alteration of the gray substance;* and it would be entirely consistent with this assumption, if it should be found that the retardation always affects the sensation of pain only, and never that of touch. It would then, according to Schiff, depend on the condition of the posterior columns, whether the sensation of touch is absolutely wanting, or appears in reduced amount, but with the normal rapidity.

The investigations of Burckhardt,[2] who has tried to measure the sensory conduction of the cord in an isolated form, are of great interest. He found that the cord conducts impressions of pain decidedly more slowly than those of touch, and suspects for this reason that the gray substance in general conducts more slowly than the white. The retardation of tactile conduction under pathological circumstances is referred by him directly to a loss of white substance (degeneration of the posterior columns); the more the gray substance is called upon to perform the function of conduction, the more slowly is the function performed. He thinks, also, that every narrowing of the gray substance—which is naturally a slow conductor—must still further retard the conduction ; the examination with the second-hand of a watch will not bring out the fact of retardation until such narrowing has taken place. As long as the gray substance is intact, the retardation can only be demonstrated by the aid of fine physiological apparatus for measurement.

The retardation of the conduction of sensation is perhaps connected with another phenomenon, usually observed in the same patients—namely, *the inability to count correctly several impressions of sensation which follow rapidly* (*e. g.*, pricks of a needle).

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[1] See Physiol. p. 294. Coroll. 3. c.

[2] Physiolog. Diagnostik der Nervenkrankheiten. Leipzig. 1875.

Persons in health are able to state without fail the number of pricks (from two to six), even when they come very close together, while patients cannot do this unless the individual impressions follow at considerable intervals. These intervals are supposed to bear a direct ratio to the degree of retardation of the conduction of sensation. This symptom would therefore seem to be also dependent on a change in the gray substance. Nor is it easy to see why the impressions are not perceived as separate, since each impression must have precisely the same obstacles to surmount.

It is more probable that this phenomenon is connected with another disturbance, which commonly accompanies it—namely, *remarkably persistent after-sensations*, following impressions of pain. The patients, when their skin is pinched or pierced with a needle, give expression to a much longer and severer pain than is usually felt by well persons. Rapidly succeeding impressions of sensation run together into one, therefore, because the new sensation coincides with the after-sensation of the one before it. The change in the cord upon which this depends cannot at present be stated with certainty. We may suppose that there are coincident changes in the posterior columns and the gray substance.

b. Exaggeration of Sensory Action.

This is one of the commonest symptoms in diseases of the cord, and may appear in various forms, viz.:

1. As *simple hyperæsthesia;* more or less increase of sensitiveness to all possible kinds of sensory impressions, which directly increase to pain. This hyperæsthesia not seldom resembles anæsthesia in its manner of appearance and distribution—in fact, it often precedes anæsthesia; thus, a hyperæsthesia in the form of a girdle may be observed above or below an anæsthetic zone, and may gradually move its position on the body, in company with the latter. Hyperæsthesia may also be confined to single denominations of sensibility (pain, sense of temperature, especially sense of cold), and may occur in connection with partial paralysis of sensation.

It is known from physiological experiments that section of the posterior columns is followed by a hyperæsthesia of the posterior half of the body, which at first increases rapidly and considerably, and afterwards very gradually diminishes and disappears;[1] and that when only one posterior column is cut, the hyperæsthesia remains confined to the same side. The hyperæsthesia increases, if the cut is carried into the lateral columns, and a part of the gray substance (Brown-Séquard); it is much less marked when the lateral or anterior columns are cut, and the posterior are left intact.

It is hard to give a decided interpretation of these facts. Tuerck and Schiff think them due to a state of irritation of the cut parts and the adjoining regions, especially the posterior columns. But the finer mechanism of the process is still uncertain, and the conducting paths are unknown. May it not be that the narrowing of the sensory conductors, produced by the section, has something to do with this result, by increasing the excitement of such conductors as remain intact?

At all events, Schiff's assumption of an irritative condition in the posterior columns is in satisfactory agreement with the fact that these hyperæsthesiæ are by far the most common in such forms of disease as we have been accustomed to ascribe to degeneration of the posterior columns. Nevertheless, it is not unlikely that there are other processes capable of occasioning hyperæsthesia, *e. g.*, implications of the nerve-roots in meningitis, etc.

2. As *paræsthesia.* Nothing is commoner than to hear patients with disease of the cord complain of abnormal sensations, which are best called *subjective sensations of touch.* Thus the feeling of furriness (Pelzigsein), numbness, crawling, tingling, etc. These sensations are referred by Schiff to a moderate excitation of the paths for the sensation of touch, lying in the posterior columns—an hypothesis which seems a little bold, in view of the fact that the posterior columns, with the exception of the root-fibres crossing them, are stated to be inexcitable. The hypothesis would have to be supported by the further one, that

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[1] See *Schiff*, Physiol. p. 274.

pathological processes are able so to change the excitability of the posterior columns that pathological stimuli will arouse sensations.

It is certainly possible that excitation of the posterior roots at their entrance may assist in producing such changes in the sensation of touch; and that a part of these modifications may depend simply upon a dulling of the sensibility (as regards touch), produced by various diseases of the cord.

Subjective sensations of temperature also occur, a feeling of burning or cold that may become very intense. These sensations are in part referred by Brown-Séquard to direct excitation of the fibres in the gray matter which conduct the sensations of temperature. Schiff, however, believes that changes in the amount of blood circulating in the skin, due to vaso-motor disturbances, may so act upon parts which are already hyperæsthetic as to produce a sensation of increase or diminution in the warmth of the skin. But this explanation can hardly suffice for all cases.

This is the place to speak of the *girdle-sensation*, that peculiar modification of subjective perception which produces the impression of having a girdle or a broad bandage tied about the trunk or limbs. This feeling, when situated at the upper part of the thorax, may be accompanied with a severe sense of pressure, and is always very troublesome to the patient. Cruveilhier described it. It may occupy various levels on the trunk, but may also attack various parts of the lower extremities, particularly in the region of the ankle and knee of one or both sides.

This sensation is probably produced by a slight excitation of the entering posterior roots in cases where the spinal affection is limited in its longitudinal extension. It usually accompanies inflammatory or other irritative conditions of the cord, and originates with the root-fibres which occupy the upper limit of the disease. But any sort of local disease of the cord and its neighboring parts, which irritates the posterior roots to a moderate extent, may produce the symptom.

3. As *pain*. This is seldom quite absent in diseases of the cord; it varies extremely in form and distribution.

Among the most characteristic are the so-called *lancinating* or *neuralgiform pains*, which are almost pathognomonic of the early stage of tabes dorsalis. These pains are usually very severe; they are either continuous, or appear periodically and under certain provocations (especially change of weather, rain, storm, snow-squalls), and are localized in a certain nerve or certain fibres of it, or certain regions of skin; they rage for a time in one place, and then leap suddenly to another, seldom remaining long in one spot. They are described as tearing, shooting, or going through like lightning; the patient feels as if a knife or a red-hot wire were thrust into his flesh, or as if certain portions of his limbs were screwed up in a vice; often the pains are localized in deep parts, as the bones, but they often invade the skin, where they are frequently connected with circumscribed hyperæsthesia. They prefer the night, and are not rarely connected with circumscript vaso-motor disturbances, or even with reflex muscular jerkings. They may occur in any nerve-region whatever, though they are certainly most common in the lower limbs and the trunk, where they often simulate intercostal neuralgia very closely; they may appear in the upper extremities, and even in the region of the trigeminus.

The origin of these pains is almost universally referred to irritation of the posterior root-fibres; their extent and location depend on the extent to which the latter are implicated. The cases of which they form a symptom are almost exclusively those of degeneration and sclerosis of the posterior columns, and, according to Charcot, of the so-called external bands solely, which contain the inner root-fibres. It is, however, also possible that irritation of the longitudinal fibres of the posterior columns, or of the gray substance, may lead to such excentric[1] pains, under pathological conditions; though usually the gray substance is only æsthesodic.

The localization of these and similar pains in the dorsal nerves and a part of the lumbar nerves produces the *girdle-pain*. This is a neuralgic pain, which may take the form of a bilateral neuralgia of the intercostal or lumbo-abdominal nerves, at various levels on the trunk, but is often confined to one side. It occurs in cases of limited irritation of the dorsal cord, and still oftener in diseases which directly irritate the sensitive roots, as especially in inflammation, caries, carcinoma of the vertebræ, etc.; it is a valuable sign of the presence of the latter, and often betrays at a very early period the beginning and the location of a severe disease, which by degrees leads to compression of the cord.

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[1] *i. e.*, originating in a central organ.

Generalized pains are not infrequently met with in the lower extremities, and in the portions of the trunk situated below the point of disease. These pains may differ greatly in degree, and are described by the patient as a more or less extensive sensation of pain, hard to describe, but exceedingly unpleasant, which is usually continuous, but from time to time undergoes exacerbations. In some the feet and lower half of the legs are the chief seat of these pains; others complain more of the back, the loins, or the thigh; very often the pains are excited or increased by spontaneous or reflex twitchings and spasms in the lower (paralyzed) extremities, or by efforts to walk. They occur in all sorts of diffuse, transverse myelitis, in compression of the cord followed by myelitis; in acute and chronic spinal meningitis, etc.

The source of these pains is not yet fully clear. The original explanation referred them to a direct irritation of the root-fibres within or without the cord; but it is probable that an irritation of the æsthesodic paths in the cord may have the same effect. Schiff, however, denies the possibility of this, believing that, in such cases, the disease always extends to the root-fibres. Many facts render it probable that pathological conditions may considerably alter the excitability of the æsthesodic substance, and it is possible that pathological irritations act differently from our coarse mechanical or electrical stimulations. Hyperæsthesia, when present, may also assist in the production of such pains.

Special notice is due to the *pain in the back*, so common in diseases of the spinal cord. It accompanies a great number of spinal diseases, assumes a great variety of forms, and is referable to a variety of causes. Thus *rheumatic* or *rheumatoid* pains, which are most frequent; they are localized in single, distinct muscles, are excited by certain movements, respiration, or pressure, and are almost always referable to the influence of cold.

Although they occur at times in well persons, they are very especially common in spinal patients, who, as a class, are very sensitive to cold, and in the latter may be excited in similar ways by a variety of influences which weaken or irritate the spinal cord, as excesses in the use of alcoholic drink or in sexual indulgence.

Hyperæsthetic pains in the back also occur, consisting of burning, tearing, or duller sensations in the skin of the back, especially between the shoulder-blades, or at certain spinous processes, which, in these cases, become extremely sensitive (spinal irritation). This pain indicates abnormal conditions of irritation and hyperæsthesia in the posterior roots and columns, and may be more or less diffuse, according to the extension of these processes. The *excentric neuralgiform pains*, which have been already mentioned, may of course also occur in the back. They are very violent, tearing, boring, localized in various spots, according to the seat of lesion, but preferring the region of the nape or the loins. Inflammations, hæmorrhages, tumors, degenerations of the cord, etc., produce these pains, and they probably point, in most cases, to pathological irritation of the root-fibres. The pain is often of much significance in *affections of the vertebræ;* it is localized in one or several spinous processes, is especially felt when pressure is made upon them, or when the spine is moved, is usually associated with excentric girdle-pains, and with a very rigid position of the spine; but the latter circumstance occurs in some other kinds of spinal pain, without lesion of the vertebræ.[1]

Pains localized in the vertebral column are best examined by pressure on the spinous processes, or by tapping them with the percussion hammer or fist, or by vigorous flexion of the spinal column, a forcible push of the head or the shoulders, etc.; the hyperæsthetic portions may also be very well ascertained by passing over them a sponge dipped in cold or hot water, or by electrical examination.

A brief notice of *headache* is necessary, which, not including accidental complications (fever, cerebral disease), is not a rare accompaniment of spinal diseases. A direct involvement of the sensitive fibres of the cervical plexus in the lesion of the cord may give rise to (occipital) pain; in like manner the trigeminus, which receives an ascending root from the cervical cord, may sympathize; finally, headaches are not seldom observed, which resemble hemicrania, and are perhaps referable to an implication of the conductors which lie in the cervical sympathetic and originate in the cervical medulla. It follows that any sort of continued and violent pain of the head is generally to be ascribed to an affection of the cervical medulla. Such pains occur in tabes, in local sclerosis, in bulbar paralysis, tumors of the cervical medulla, etc. (*i. e.*, in cases of disease of the cord).

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[1] Compare also *A. Mayer*, Die Bedeutung des Rückenschmerzes bei Erkrankungen des Rückenmarks und der umgebenden Theile. Arch. der Heilk. I. p. 349. 1860.

2. *Disturbances of Motility.*

These are the most common, and in many cases the predominant and most troublesome symptoms of spinal cord diseases. They deserve a most attentive study in all cases.

The proper methods for *examining the motor apparatus* have been fully explained by me in Vol. XI. of this Cyclopædia, p. 267, and I would refer to the statement there made. It cannot too often be repeated, that the examination of these points should be as thorough and general as possible in all cases; in many cases of difficult diagnosis this furnishes the only possible means of attaining an exact idea of the disease, and it is only by this path that we can ever hope to reach a clearer definition of diseases than we now possess.

a. Diminution of Motility—Weakness and Paralysis.

All degrees of "palsy," from the slightest paresis to complete paralysis, occur in diseases of the cord; and as to the situation any part may be affected, though by no means with equal frequency.

In the earlier stages the patients complain of being quickly fatigued, of diminished power of performance and endurance in their limbs, then of a slight weakness and incertitude in executing certain movements, perceived only by themselves, and at last a slight dragging of the legs is observed. In these early stages it is often especially striking to observe the inability to stand still for any long time.

By degrees the symptoms of weakness become plainer; it becomes harder and harder for patients to mount on a chair or ascend stairs; every little impediment in their way annoys them and detains them; their powers grow continually weaker, short distances exhaust them completely, they have to stop or sit down at every other step.

Thus it goes on till palsy is complete, with absolute inability to move the muscles; it may be weeks, months, and years before this point is reached.

But, on the other hand, the paralysis may occur almost suddenly, becoming complete in a few minutes or hours; bed-ridden patients often observe, at the moment when they wish to make use of their limbs, that they are more or less completely paralyzed, so stealthily and rapidly may the palsy develop itself. This point depends on the nature of the disease of the cord which causes the paralysis.

Our observations upon this matter are defective enough; but as far as they go, they allow us to associate spinal paralytic symptoms in the first place with affections of the anterior half of the cord; it seems from pathological observation that the worst disturbances of voluntary motion originate in the lateral columns and the anterior cornua. We are not yet certain as to the part played in man by the anterior columns proper. It is clear that the causal lesion of paralysis may have various locations; in the anterior roots, within or without the cord, in the large (motor) ganglion cells of the anterior cornua and their immediate offshoots, or finally, in the paths which lead up in the antero-lateral columns to the brain. The lesion may, further, be circumscript, or may extend over a great part of the length of the cord.

The nature and the distribution of the palsy are not very characteristic by themselves, but their combinations with other symptoms give many points which assist in an accurate localization. Thus the presence or absence of reflex actions, secondary muscular atrophy, muscular tension and contracture, changes in electrical excitability, etc., furnish very important landmarks of the seat of disease, and it will be proper here to mention briefly some of these points.

Paralysis rapidly followed by a marked degree of atrophy and by the reaction characteristic of degeneration (Entartungsreaction)[1] points to disease of the anterior roots (rarely), or of the gray anterior cornua (more frequently). In this case all reflex actions are absent.

Paralysis with tension and contracture of muscles, without atrophy, is very probably due to an affection of the lateral columns.

Paralysis without loss of reflex function and without atrophy points to an affection of the paths which ascend to the brain, outside of the gray substance, or at least outside of the ganglia of the anterior cornua. Such are mostly cases of circumscript disturbances of conduction, the end of the cord below the lesion remaining intact.

Paralysis with trophic disturbances gives room for suspecting an affection of the gray substance, since primary affections of the roots are very rare.

Very extensive palsy with much atrophy, the reaction of degeneration, absence of reflex acts, points to a widely diffused lesion of the anterior gray substance.

Paralysis in the districts of certain pairs of roots (*e. g.*, in those of the upper extremities alone, or both crural nerves, etc.), points to a strictly localized affection of roots or lesion of the gray anterior cornua.

Of course these statements are not by any means exhaustive, and give only general assistance; the difficulty of making the distinctions is at present very great; in many cases a variety of other circumstances (spasms, anæsthesia, pains, palsy of the bladder, etc.) may assist the judgment, but in other cases they only add to the difficulty. Such combinations are very common, and are extremely various, especially in the different forms of myelitis.

The conclusions which are formed regarding the *nature* of the lesion in the cord are far less certain than those relating to its *place*. The diagnosis is commonly established by a consideration of the symptoms as a total.

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[1] Volume XI. p. 426.

In respect to the *extension* of the disease, a few remarks remain to be made.

By far the commonest case is that in which the lower extremities, usually both together or nearly so, are attacked by the paresis or paralysis, which gradually ascends, reaching successively the trunk and the upper limbs. In fact, *paraplegia* is so characteristic a form of spinal palsy, that when it occurs we always think first of a spinal affection. (A paresis occurring in this form may be designated as paraparesis.) Paralysis of both lower extremities and the trunk to various heights, accompanied by disturbances of sensibility, palsy of the bladder and rectum, and bed-sores—such is the usual form; but the latter symptoms may be entirely wanting.

The most frequent causes of paraplegia are affections which involve the thickness of the cord, or complete compression from vertebral caries, tumors, etc.; but paraplegia occurs also in affections which are strictly localized in the motor apparatus, such as spinal palsy of children, hemorrhage in the gray anterior cornua, etc.

If the upper extremities, and finally the muscles of respiration, are also invaded, the affection takes the form of *universal spinal paralysis*, which is observed in various spinal affections, described in the special part of this work.

If only the two upper extremities are attacked by palsy, the legs remaining free, we have *paraplegia brachialis* or *cervicalis;* a rather rare form. It occurs in connection with processes which affect in an isolated manner the anterior roots of the cervical enlargement, or in strictly circumscribed lesions of the anterior cornua in the cervical enlargement (as in spinal palsy of children, progressive muscular atrophy, perhaps also lead palsy [?]). In disease of the white columns it is rare that an isolated affection of the paths for the upper extremities occurs.

Hemiplegia spinalis (Brown-Séquard) is the name given to palsy of an arm and a leg of the same side, originating in a spinal affection; the face is not attacked. It occurs in unilateral disease or injury of the cord, in which case the motor palsy is on the same side as the lesion of the cord, while sensory palsy exists on the other side. If this palsy is confined to one lower extremity, it is called *hemiparaplegia spinalis*. For further remarks see below, in the section upon Unilateral Lesion of the Cord.

Finally, *partial paralyses* of spinal origin are frequent. They may be limited to a single extremity, single groups of muscles and nerve-territories, or even single muscles; this depends entirely on the nature and distribution of the lesion in the cord. Such partial palsies are usually due to quite circumscribed local lesions, which show little tendency to spread; small hemorrhages in the medulla, circumscribed myelitic foci in the gray substance, little islands of sclerosis, etc. It is often hard, or even impossible, to distinguish these from circumscript affections of the roots or other peripheral palsies.

b. Imperfect Co-ordination of Movements—Ataxia.

This peculiar and frequent disturbance of movement has been made the subject of numerous debates within the past ten or twenty years, since Duchenne introduced the term "ataxie locomotrice" into nosology.

Ataxia is characterized by inability to make combined or complicated movements with certainty and exactitude, or even (in advanced cases) to make them at all, while the simple individual motions and the gross force of the muscles are normal, or but little impaired.

This disturbance is most marked in *standing* and *walking*. A patient who presents the characteristic signs cannot perform these acts with security; in walking, he brings his foot down with a stamp, the motions of his legs are exaggerated, wild, jerking, the movements are various, impulsive, often made in the wrong direction and with an unsuitable degree of force.

It soon becomes necessary to exercise an increased control with the eyes over the movements. The patient has to keep his eyes on his feet and on the ground while walking; in the dark, or with closed eyes, his uncertainty increases considerably, especially when there exists an impairment of the sensibility of the legs. It soon becomes impossible to walk without the aid of a cane, or a pair of sticks; finally this also, and even standing, is beyond his power.

While lying down, all the simple movements are at first performed with ease and certainty, and even with normal force; but a distinct failure of force, and, still more, of endurance, is usually seen at an early stage. All complicated movements, on the contrary (describing a circle or other figure with the tip of the toes, touching objects with the toes, etc.), are more or less interfered with, even in the lying posture, by the zigzag movements of the leg. This at last extends to the simple movements; the leg is jerked hither and thither, away from the intended line of action, or it falls back upon the bed in a spot different from that intended.

In the severest forms of ataxia, every attempt at innervation puts a great number of muscles in action; the limbs are thrown about irregularly, and perform clonic shaking motions which are beyond the control of the will. These motions may extend from one to the other leg, and in severe cases even to the trunk and arms; they cease as soon as voluntary acts are not attempted.

In the arms and hands we observe the same order of progress; all the finer complicated movements become uncertain, clumsy, sprawling, and at last quite beyond the power of the patient to execute. When he tries to seize an object, he passes by it, spreads his fingers at the moment when he should grasp, moves his hand forwards in irregular zig-zags, and reaches his object with difficulty and after many efforts. He cannot carry his food to his mouth, spills the contents of his spoon and glass, thrusts these objects into his face, etc. Buttoning the clothes, sewing, writing, playing the piano, soon become impossible from the interference of the involuntary motions; in severe cases a shaking and sprawling accompany every attempt to move, and put the patient in a condition of complete helplessness.

But the gross force is very often retained to a great extent and for a long time in the arms, the simple movements of extension and flexion being performed quite well; if a hand is offered, the patient presses it powerfully, and is able to make very energetic resistance to passive movements.

In rare cases the same disturbance of movement seems to extend to the *speech*, and even to the motions of the *eyes*.

If the phenomenon is closely examined, it is at once evident that the motor disturbance is of a peculiar kind. The simple motor conduction is not disturbed; it is entirely possible to perform any simple motion; the force of the muscles is often retained for a long time, or only a little diminished; *the case cannot, therefore, be one of real palsy*, however helpless the patient may often be made by these disturbances. There is rather a deficient harmony among the impulses to motion which are requisite for every combined and associated movement. We may therefore give the following definition: *Ataxia is the disturbance of movement, produced by defective co-ordination of the latter.* Wherever a co-ordination of several muscles is requisite to the production of a certain movement, this symptom appears, and is distinct in proportion as the desired movement is complicated.

The *manner in which ataxia may be produced* appears from that which has already been stated (see p. 39 et seq.) concerning the co-ordination of movements—namely,

a. By abnormal extension of the motor innervation to too many or too few muscles, so that in some cases more, in others fewer than are normally required, are put in use for the attainment of a definite object of motion.

b. By abnormal strength of the innervation sent to each muscle in the case of a complicated movement.

A division of these disturbances into *ataxia proper* (cases under *a*) and *disturbances of innervation* (under *b*), as proposed by Cyon,[1] is impracticable. The effect of both disturbances upon the visible movement is plainly the same. And since, at any rate, the two functions—the choice of the muscles to be innervated, and the strength of the single innervations—are simultaneously executed by the same apparatus (that for co-ordination), the disturbances of this apparatus will always affect both functions more or less.

Exactly how these disturbances come to pass is hard to say; irritative and inflammatory processes in the apparatus for co-ordination may sometimes be the cause.

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[1] Zur Lehre von der Tabes dorsualis. Berlin. 1867.

It has been stated above, that the proper centres of co-ordination do *not* lie in the spinal cord; that at all events they are by no means to be demonstrated in it.

By this one fact, the hypothesis of Brown-Séquard, Jaccoud, Cyon, and others is rendered very improbable; these authors believe that in spinal affections ataxia originates in a disturbance of the reflex function, because, under normal circumstances, co-ordination is effected reflexly within the cord, in the gray substance. Although there are a few motor acts, such as standing and walking, in which it is impossible entirely to deny the co-operation of reflex processes; although, moreover, the absence of the tendon-reflex in tabes (recently discovered by Westphal, and confirmed by myself) might be employed to assist this position, yet a closer consideration shows that it is entirely untenable. The subject cannot be further discussed here.

In ataxias originating in spinal disease only those conductors can be disturbed whose assistance is required in producing co-ordination. Such paths are either

Sensory paths (for the cutaneous or muscular sensation, etc.), which are of use in superintending the movements and in maintaining the equilibrium of the body; or

Those *motor paths* which convey the impulses from the centres of co-ordination to the motor roots; these paths are probably distinct from the simple paths for motor conduction, which furnish a *direct* connection between the voluntary centres and the muscles; they form a sort of side-channel.

Ataxia in diseases of the spinal cord must therefore be either *sensory* (caused by disturbance of the centripetal paths) or *motor* (caused by disturbance of the centrifugal paths[1]).

The existence of these two forms, and the evidences for supposing their existence in various spinal diseases, form the nucleus for the recent theoretic dispute about the existence of spinal ataxia of movement.

A very exquisite ataxia is found in various spinal diseases, as sclérose en plaques, and more especially in tabes dorsalis (ataxie locomotrice progressive, gray degeneration of the posterior columns). The strife is very hot upon the latter point.

While authors like Friedreich, Spaeth, Niemeyer, Topinard,

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[1] *Central* ataxia, caused by disease of the centres of co-ordination, may be placed in a class distinct from these forms.

Finkelnburg, and others, have supported the theory of motor ataxia, others, as Axenfeld, Landry, Leyden, Ruehle, Clifford Allbutt, have tried to base ataxia upon sensory disturbances. The view of Leyden is at first very plausible; it has been thoroughly stated in several works,[1] and has gained a very large number of supporters. We will briefly inquire whether the reasons brought forward in support of this view are sufficient or not.

Leyden's theory of ataxia may be stated as follows: Co-ordination of movements is rendered possible, and is effected through sensibility; suspension of the sensibility (of the skin, joints, muscles, etc.) suspends co-ordination; gray degeneration of the posterior columns is accompanied by disturbance of sensibility, along with the ataxia; we are acquainted with no other than sensory functions in the posterior columns; consequently, ataxia is a result of disturbance of sensibility.

First, we cannot consider the experimental proof of this theory as very fortunate; we do not wish to endorse Cyon's violent criticism, but we are forced with him to draw from Leyden's first series of experiments the conclusion, "that the disturbance in muscular function which follows section of the posterior roots has nothing in common with what we are accustomed to call disturbance of co-ordination." And the second series of experiments on frogs,[2] with section of the posterior regions of the cord, cannot prove anything in regard to the present question, not to speak of the great complication of the circumstances; it proves at most that when certain portions of the cord are cut, disturbances of sensibility and of co-ordination appear simultaneously. It is impossible to draw from these experiments a conclusion as to the dependence of the latter upon the former.

The demonstration from pathological cases is based on the fact that in not a few cases of ataxia there exist various degrees of disturbance of sensibility, and especially disturbance of the so-called muscular sense. This also proves nothing of itself; it shows, at the most, that in gray degeneration of the posterior columns sensory and co-ordinatory paths are attacked together.

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[1] Die graue Degeneration der Hinterstränge des R.-M. 1863.—Zur grauen Degeneration der hinteren Rückenmarksstränge. Virch. Arch. Bd. 40. 1867.—Ueber Muskelsinn und Ataxie. Virch. Arch. Bd. 47. p. 321. 1869.

[2] Virchow's Arch. Bd. 40. p. 198.

A positive argument against this view is, however, found in the *disproportion between the intensity of the sensory disturbance and the ataxia;* there are cases of severe ataxia with slight disturbance of sensibility, and of severe disturbance of sensibility with slight ataxia; they are not rare in any considerable series of cases.

Again, the existence of *ataxia in a high degree without any disturbance of sensibility* militates against this view. Friedreich[1] has published such cases. Leyden does not fully admit their existence. I myself, however, have very recently examined two cases of this sort most carefully in regard to this point, and have found severe ataxia associated with a sensibility completely intact in every point (touch, temperature, pressure, pain, sense of tickling, muscular feeling, feeling of position of limbs, of passive movements, etc.), so that the existence of such cases is proved to my mind beyond the possibility of doubt.

Again, the occurrence of *severe anæsthesia without ataxia* does not harmonize with such a view. Literature is not wanting in cases in which there was anæsthesia of the legs, without ataxia, from one or another cause. In the printed accounts of cases of unilateral lesion, moreover, I have found that ataxia has never been observed in the anæsthetic leg. These cases, however, may be met with the statement that only cutaneous anæsthesia was present, while the muscular sense was intact.

The only decisive case would be that of *complete spinal anæsthesia* (involving the skin, joints, muscles, etc.) *without ataxia. Such a case exists.* It was repeatedly examined by various trustworthy observers, with special regard to the question in hand; an autopsy was had, and the state of the cord was given with great accuracy. This is the case of Remigius Leins, first published in Spaeth's work;[2] the autopsy was fully described by Schueppel.[3] Its importance obliges us to give a brief account of it.

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[1] Virchow's Arch. Bd. 26 and 27. 1863.

[2] Beitr. zur Lehre von der Tabes dorsualis. Tübingen. 1864.

[3] Ueber einen Fall von allgemeiner Anästhesie. Arch. d. Heilk. XV. 1874. p. 44.

Remigius Leins, aged forty-two, in the year 1862, has suffered for twenty years with anæsthesia of the hands and arms, which rapidly became severe; for six years similar troubles have existed in the lower extremities. Present condition: upper extremities wholly anæsthetic; on the soles of the feet the sensations of touch, pressure, and pain are entirely extinct, and in the legs considerably diminished. He falls when his eyes are closed. In the dark, when in bed, he feels as if floating in the air, as the anæsthesia extends to the trunk.

March, 1864.—Sense of pressure in the upper extremity, and the sense of force, entirely extinct. *Sense of position of the upper extremity and of passive movements of the latter completely extinct. Movements of the upper extremities powerful and perfectly correct;* the patient eats alone, dresses himself, etc., as far as he can direct his acts with his sight. When the eyes are closed the arms are moved nearly like those of a blind man. In the *lower extremity,* besides the cutaneous anæsthesia, there is *complete loss of the sense of passive movements and of the position of limbs.* In spite of this the patient *can walk without support, quite fast and securely, for a good distance. If he is asked to raise his foot to a given height while his eyes are shut, he accomplishes the act by a perfectly quiet and suitable motion.*

June, 1872.—Sensibility continues the same. When the eyes are shut he has no idea at all of the position of his limbs, and, if standing, falls. He can still walk, clumsily, but not atactically. He can perform all desired actions with his arms, as long as he can see them.

Death, May, 1873.

Autopsy.—A cavity in the entire length of the cord, from the level of the first cervical nerve to the first lumbar. Posterior columns in the lower half of the cervical medulla wholly destroyed and gone; above, gray degeneration; in the dorsal part slight atrophy and increase of connective tissue; the lumbar part normal. Anterior columns everywhere quite uninjured and normal. Anterior commissure, from the second cervical to the twelfth dorsal nerve, completely destroyed. Lateral columns in the same regions sclerotic in the neighborhood of the posterior cornua. Gray substance mostly implicated in the cavity; gray commissure and posterior cornua almost wholly destroyed in the entire cervical and dorsal medulla; anterior cornua almost entire, and only in the cervical region reduced to a small size; a lateral strip of gray substance is also retained everywhere. Anterior roots normal. Posterior roots, from the third to the eighth cervical nerves, in a state of complete connective-tissue degeneration,[1] and, to the end of the dorsal medulla, more or less atrophied. Lumbar part, with its roots, normal; etc.

This case is perfectly clear and convincing; in my opinion it entirely overthrows Leyden's theory. If the maintenance of sensibility were a necessary condition of co-ordination of movements, the extreme of ataxia ought to have accompanied this absolute anæsthesia; but *there was no trace of ataxia.*

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[1] This fact is also very decisive against the reflex theory of Brown-Séquard and Cyon.

It results without question from this, that the retention of sensibility is not necessary, in order that co-ordinate movements shall be performed; it may be necessary in acquiring the faculty of doing them, and without doubt it is of great importance in maintaining equilibrium, but it is *not indispensable in performing co-ordinate movements which have been learnt.* Loss of sensibility, therefore, can in no way interfere with these movements, when once acquired by practice.

It seems to us, therefore, quite unjustifiable, upon scientific grounds, to make a disturbance of sensibility responsible for ataxia, when both occur together. It must rather be believed, that there exist special co-ordinatory paths in the cord, which are attacked in tabes dorsalis and cognate affections, when the symptoms of ataxia appear.

In the present state of our knowledge, therefore, *we have no right to assume any other than a motor ataxia in tabes.*

In examining the question whether a motor ataxia can be objectively distinguished from the sensory form, this will become still plainer; it will also be seen whether, and how far, the assumption of a sensory ataxia is justified.

A *purely motor form* of ataxia is to be assumed when the sensory apparatus is entirely normal (sensibility, muscular sensation, vision). If sensibility (in its widest sense) is perfect, and the movements are still atactic, it must be that the cause lies only in the apparatus for co-ordination, and not in the accessory sensory apparatus. We have above shown that such cases exist beyond a doubt.

We know further, from two series of observations, mutually confirmatory, that the retention of a single sensory control-apparatus is sufficient to render possible a perfect co-ordination of movements, if only the apparatus for co-ordination is itself normal. For (1) blind persons, or well persons with shut eyes, show no trace of ataxia, and (2) anæsthetic patients, even when the cutaneous and the muscular sensibility, etc., are completely extinct, show no trace of ataxia as long as their eyes are open and they can control their movements by vision; this is an incontrovertible inference from the Spaeth-Schueppel case.

Hence we may conclude that a case of ataxia, even when only *one* sensory control-apparatus remains in activity, must be of a purely motor nature. This is the case when an anæsthetic patient makes atactic movements with open eyes, or when, with normal sensibility, the eyes being blind or shut, ataxia is present. Such cases are indeed not wanting; they form the great majority of tabetic diseases; they must, therefore, be instances of motor ataxia.

It is harder to characterize *sensory ataxia;* and it is questionable whether that which is commonly called ataxia ever originates in disturbance of the sensory control.

If, in spinal affections, all voluntary, complex movements, such as are acquired by practice, are well performed, and if no disturbances occur until movements are attempted, for which a sensory control is indispensable (*e. g.*, maintaining equilibrium, standing upright, etc.), then there will be a certain propriety in speaking of sensory ataxia. It will be recognized by observing that disturbances of motion are absent, so long as even one sensory control-apparatus is in action, but make their appearance when, one apparatus being already impaired, the other (intact) apparatus is excluded; thus, when a blind man becomes anæsthetic, or—to choose a more likely example—when an anæsthetic patient closes his eyes. In these cases, considerable disturbances of motion will inevitably occur. It seems to us, however, extremely questionable whether these disturbances show an agreement with or even a resemblance to that which we call ataxia.

When a patient whose hands are anæsthetic shuts his eyes, he can no longer hold firmly a needle, a button, or similar objects; he cannot fasten his clothes, etc.; the things fall from his hand, he does not complete the motions, he performs them falsely —but he does not become atactic. The movements are correctly willed, and probably also correctly executed; but the patient is no longer able to judge whether the end is attained; the movements are therefore often carried beyond the point desired, or they fail to reach it—but they do not become properly atactic. It is the same as when an object is held before a blindfolded person, and he is asked to seize it; he will make the most unsuitable movements, but they will not be atactic.

If the feet are anæsthetic, and the patient shuts his eyes while standing, he will fall immediately, because he has no oversight ("Controle") as to whether the voluntary efforts for the purpose of retaining the equilibrium are sufficient or insufficient. In lesser degrees of anæsthesia, tottering, at least, will occur, because an adequately strong sensory impression is not made by any minute deflection of the body. Walking with closed eyes becomes uncertain, tottering, even impossible; but it is not at all necessary that a proper ataxia should exist. This also results from the case of Spaeth and Schueppel.

The intentions of the will may be formed and executed quite properly, but they are incorrect with respect to the desired object, being too great or too small, because the patient is deprived of a standard by which to measure them. *In this case, therefore, the purposes of the will and the voluntary impulses to movement are false, but they are correctly carried out, while in ataxia proper the purposes of the will are correct, but are falsely carried out.*

The notion of sensory ataxia is, therefore, only admissible in case the motor impulses which maintain the equilibrium are produced quite involuntarily by the action of centripetal excitement on motor paths (in the thalamus opticus, the corpora quadrigemina, or the cerebellum), that is, in apparatus which is usually called co-ordinatory. But the symptoms of such cases are certainly essentially different from those of motor ataxia.

It seems to me, however, much more desirable *to separate the processes which serve to maintain the equilibrium and position in space from the processes of co-ordination proper of the (voluntary) movements;* this will certainly conduce to clearness in the question of ataxia. The discovery by Goltz of the different centres for equilibrium (lobi optici) and for locomotion of the body (cerebellum) is a weighty argument for this separation. Of course, the processes of movement which contribute to the maintenance of equilibrium, etc., equally require the apparatus of co-ordination in order to act normally, and this is put in action by impulses from the centres for equilibrium, as it is by the centres of volition in voluntary actions. The centres of equilibrium, therefore, and those of volition may be considered as standing in a similar relation to the apparatus for co-ordination.

It follows directly that a disturbance of the centres of equilibrium does not at all necessitate a disturbance of co-ordination of voluntary movements; also that a disturbance of the sensory excitations which put the centre of equilibrium in action is not necessarily followed by a disturbance of co-ordination, but that, on the other hand, every disturbance of the apparatus of co-ordination will more or less impair the performance of the movements necessary for equilibrium. It will be well in future to apply the test separately in both directions in these cases of spinal disease.

This brings before us the consideration of another motor symptom, which is usually placed in the most intimate connection with ataxia, namely, *tottering and falling when the eyes are shut*, a symptom to which, under the name of the Brach-Romberg symptom, too much importance has certainly been attached.

It is easy to prove that many patients with disease of the cord (especially tabetic patients, with a more or less marked ataxia and disturbance of sensibility), who are still able to walk and stand quite tolerably with open eyes, begin immediately to totter as soon as their eyes are shut; the tottering becomes worse and worse, and in severe cases the patient presently falls. This tottering is most plain when the eyes are closed, and the patient is made to stand with his feet together. The intensity of this disturbance usually seems to be directly proportionate to the degree of ataxia, but this is only apparently true.

This phenomenon is evidently due to a disturbance of equilibrium and of the power of maintaining position in space. We have already shown that these cannot be maintained without a continued sensory control (principally furnished by the sensory impressions in the lower limbs and by the sense of sight). If *one* part of this sensory control be taken away by shutting the eyes, the maintenance of equilibrium and position in space will be difficult, in proportion as the *other* factor is already impaired, that is, corresponding with the existing disturbance of sensibility.

In fact, this symptom also is found chiefly or exclusively in cases of marked disturbance of sensibility of the lower limbs; in complete anæsthesia the patients fall quickly when their eyes are closed. *This phenomenon, therefore, is simply an indication that the sensory control exercised by the soles of the feet, the joints, and the muscles is insufficient.* This is in harmony with Benedikt's statement, that in a great many instances of difficulty in standing with closed eyes, he has never seen one where there was not a disturbance of the muscular consciousness. But, on the other hand, there are some quite severe cases of ataxy (as I have seen most clearly demonstrated), in which the tottering with closed eyes is absent or scarcely exists, when the sensory control from the skin, the muscles, etc., is perfectly intact, *i. e.*, when there is no disturbance of sensibility. It is well, however, to remember that in atactic persons this symptom must be more marked, since the movements which serve to maintain equilibrium are inco-ordinate, so that they usually totter very perceptibly, even when their eyes are open.

It should here be added, that in many atactic patients the want of co-ordination increases decidedly, the movements become much more excessive and ungovernable, when the eyes are shut. This proves no more than that the control by the eyes may furnish a means of partially compensating for the disturbance of co-ordination, so that, as in the process of learning how to co-ordinate, a continuous influence upon the centres for this function may be exercised; if this influence ceases (when the eyes are shut) the disturbance of co-ordination returns in its full force.

This may be the reason why atactic patients, without disturbance of sensibility, occasionally totter a little when their eyes are shut, because the muscular actions that are called into use in supporting the equilibrium, already atactic, are no longer under the direction and control of the sense of sight.

These symptoms, however, are always much more marked in cases where there is impairment of the sensibility, especially of the so-called muscular sense; in this case the movements become excessive and entirely irregular, because when the eyes are closed the sensory control entirely ceases, and to the existent disturbance of co-ordination there is added an uncertainty respecting the extent of the voluntary impulse required, the standard for estimating which has been lost. In such cases the ataxia increases considerably when the eyes are shut, while in atactic persons who retain their sensibility perfectly no increase of ataxia worth mentioning occurs, the sensory control afforded by the skin and muscles being complete and adequate.

This entire series of symptoms is only a proof that the disturbance of co-ordination may be partially made good by sensory control exercised through the vision.

We have to add, finally, that in a few cases it has been observed that atactic persons who were completely blind, but were able to stand, also experienced a distinct increase of tottering when they shut their eyes. It is clear that this cannot be due to any further diminution of the sensory control of the eyes; but it is hard to find any explanation for this singular phenomenon. The most obvious conclusion is, that the phenomenon is a psychical one. Might a sudden calling off of the attention cause an increase of uncertainty in the legs? Or may it be that a new act of motor innervation can send impulses to the co-ordinatory apparatus, which increase the existent disturbances in the latter? At present we do not know.

The preceding appears to furnish a sufficient demonstration of the fact that the chief form of ataxia in spinal affections is of a motor nature. There must, therefore, exist in the spinal cord special centrifugal fibres, employed for co-ordination (Spaeth), and ataxia in a spinal affection occurs only when these fibres are attacked. Their site is entirely unknown.

Most observers place them in the white posterior columns, because the autopsy of atactic patients usually shows gray degeneration of the posterior columns. If it were proved to a certainty that in such patients the posterior columns were *exclusively* attacked, we might regard this as demonstrated. But this is not yet proved; it is even probable that in such patients there is a more or less considerable implication of the gray substance and the lateral columns, as a rule.

Further, the case of Spaeth and Schueppel is rather decisive against the localization of the co-ordinatory paths in the posterior columns, although the duration of the case was such that a compensatory conduction might be thought possible.

Where, then, are we to look for these co-ordinatory paths? In the gray substance? In the antero-lateral columns? The experiments of Brown-Séquard, who was enabled to produce ataxia in birds by lesion of the gray substance of the ventriculus lumbalis, point rather to the gray substance. On the other hand, according to the experiments of Woroschiloff, the co-ordinatory paths lie (in rabbits) in the lateral columns, in their innermost part, in the sinus between the anterior and posterior columns. At present the question is not settled in the case of man; further exact investigations are necessary to bring it to a solution; perhaps we may gradually approach a conclusion by careful comparisons of suitable cases of spinal sclerosis in patches. Till then, our first thought in ataxia will be of a disease of the posterior columns, and especially (as would appear from Charcot's latest statements[1]) the lateral portions of these columns, adjacent to the gray substance, called the "région des bandelettes externes," region of the inner bundles of root-fibres.

c. Various Characteristic Gaits in Spinal Disease.

The form of the lesion may often be recognized by the patient's characteristic gait, as soon as he enters the room. I believe that for practical purposes it is sufficient to distinguish the following leading varieties, which may be distinctly separated:

1. The *paretic* and *paralytic gait*—caused by a more or less extensive palsy of the lower extremities. The gait is shuffling, the tip of the foot drags on the ground, the fore-part of the foot hangs down, the sole is planted awkwardly, usually with the outer edge first; the knee is raised high, or is drawn after the patient in an extended position; a certain stiffness of the legs is often remarked. The patient walks with one or two canes, or is supported by crutches or guides; he totters but little, stands quietly and securely, and when left alone simply sinks to the ground, in most cases. The manner of walking differs somewhat, according to the distribution of the palsy among the groups of muscles; if the whole leg is palsied, it is different from

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[1] *Charcot*, Leçons sur les maladies du système nerveux. II. Série. 1. fasc. Paris. 1873.

what it is in palsy of the lower half of the leg; in the latter case it is waddling, and especially characteristic.

2. The *atactic walk*—caused by disturbance of co-ordination in the legs. It is characterized by irregular hurling movements; the point of the foot is thrown forward and outward with force; the heel is brought down with a stamp, the leg stiff at the knee. The patient's eyes are continually on the ground. The gait is tottering, staggering, or even reeling from side to side; the movements are hasty, spasmodic, quite unequal; in turning about, especially, there is great uncertainty, and danger of falling. In severe cases the patient falls after a few steps.

3. The *stiff, spastic walk*—caused by reflex muscular contractions or tension associated with paresis of the leg. This gives rise to a very peculiar and characteristic walk; the legs are somewhat dragged, the feet seem to cleave to the ground, the tips of the feet find an obstacle in every inequality of the ground; every step is accompanied by a peculiar hopping elevation of the whole body, dependent on a reflex contraction of the calf; the patient immediately gets upon his toes, and slips forward on them, showing a tendency to fall forward. The legs are close together, held stiffly, the knees somewhat depressed forward, the upper part of the body slightly bent forward. There is no throwing about of the feet. This gait depends on muscular tension and reflex contractions in the various groups of muscles, which are set in activity during the process of walking.

These forms may be more or less distinct; there occur transitional forms of a mixed character; but not every patient with spinal cord disease has a characteristic gait.

d. Increase of Motility—Spasm.

Motor symptoms of irritation are among the commonest of spinal symptoms; they occur in a great variety of forms.

The simplest form is without doubt the so-called muscular tension. In this case the muscles (which are usually in a condition of greater or less paresis) are in a state of moderate tension or contracture, which makes the execution of passive movements quite difficult. This tension often occurs at the moment when a passive movement is being performed, especially if it is done rather quickly; there then follows a jerking, impulsive resistance, which enables us to recognize with ease the slighter forms of the disturbance. The voluntary movements, also, are more or less obstructed and clumsy, are performed as if the patient were in a semi-fluid medium, and require the expenditure of an abnormal amount of force.

It is easy to show that these tensions in passive motion occur chiefly in the muscles which are stretched and pulled on by the movement; thus, in passive extension, the flexors, and *vice versa.* The tension seems here to be reflex in character, and is probably connected with the abnormal tendon-reflex action, to be mentioned later.

In severer cases the movements grow more and more stiff, the resistances greater, and a condition may finally exist which resembles the flexibilitas cerea of catalepsy. The contraction is not always equally distributed through the entire muscle; in some muscles it may be only partial, forming knots or lumps.

A simple exaggeration of this state doubtless constitutes what is called *stiffness of the muscles, rigor.* The muscles are stiff and stark, much swollen, and firmly resistant to the touch; on pressure, they are usually very painful; both active and passive movements are extremely difficult. The extensor muscles are usually the principal ones affected; very often also the muscles of the nape and back (torticollis).

In the worst cases, decided *contractures* occur, which may be limited to a few muscles or groups of muscles, but often attack many in a variety of places. Sometimes the flexors, and sometimes the extensors are more affected, hence the difference of posture in the different cases.

I do not now refer to the so-called paralytic contractures,[1] which are certainly nothing uncommon, even in spinal diseases, but exclusively to neuropathic contractures, which owe their origin to abnormal states of irritation in the cord. The muscles are much shortened, their tendons quite prominent, and passive movements are entirely impracticable. If energetic attempts to perform them are made, vigorous clonic contractions of the muscles manipulated are often produced, or the contracture is increased, leading to a momentary tetanic stiffness of the whole limb.

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[1] Vol. XI. p. 379.

It would be very difficult, at the present time, to refer all these conditions of irritation to pathological changes in certain portions of the cord. But there is no doubt that there are two ways in which muscular tension and contractures may come to pass; first, a *reflex* origin may be conceived, which is doubtless the chief element in cases where sensory phenomena are prominent, in diseases of the meninges, the posterior roots, the posterior columns, the reflective gray matter, etc.; such reflex muscular contractions are said to attack by preference the flexors.

On the other hand, these symptoms may originate through *direct irritation of the motor parts* of the cord. The seat and the character of this irritation are hardly known; a direct irritation of the anterior roots is possible; according to Charcot's later observations, sclerosis of the lateral columns is a very common cause of such motor symptoms of irritation; it is not at all known how far the gray substance might produce them. In such cases of direct irritation, at least in the lower extremities, the extensors are said to be principally affected.

In respect to the exact pathogenesis of these motor symptoms of irritation, we know only this, that they chiefly occur in acute or chronic inflammatory conditions of the cord and its membranes, in the various forms of meningitis and myelitis, in many cases of multiple sclerosis, paraplegia after acute sickness, etc.

We know still less about one of the severest forms of spinal spasm, namely, tetanus, and its pathogenesis. This is a powerful tonic spasm of almost the entire muscular system; the spasm occurs in paroxysms, which are brought on, or increased, by reflex action, but continues in the intervals in the form of a moderate degree of rigidity. Tetanus is probably caused by an (inflammatory or toxic) affection of the gray substance, which enormously increases the reflex excitability. A like condition exists in meningitis spinalis.[1]

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[1] See Volume XII.

The attacks of so-called *tetany* have a remote resemblance to tetanus. By this we understand attacks of tonic spasms, occurring in regular paroxysms, and affecting the extremities chiefly. It is probably of spinal origin.[1]

Among the forms of *clonic spasm*, in spinal disease, tremor, in the first place, is not rare; either permanent or transitory, accompanying certain movements, following exhaustion, etc. Its manner of origin is as yet quite unknown; one is naturally inclined to refer it to the gray substance.

A higher degree of tremor is present in *shaking*, which accompanies and disturbs all voluntary movements in multiple spinal sclerosis; a sort of greater tremor, which begins whenever a voluntary innervation occurs, and may perhaps be regarded as a very severe degree of ataxia, although, as it appears, it is essentially distinct from the latter. We have not yet succeeded in forming an exact pathogenesis of this symptom; and we may say the same of that characteristic form of tremor, most marked during repose, which is the essential feature of *paralysis agitans*.

Few of the other clonic spasms can be referred with probability to the cord; those of single muscles, or groups of muscles, have seldom been derived from the cord, and general convulsions, as occurring in epilepsy, eclampsia, uræmia, etc., have commonly been ascribed to the medulla oblongata.

A few special cases remain to be described. Among these is a peculiar form of clonic spasm in the lower extremity, which appears in various lesions of the cord, and runs an extremely characteristic course. In its lightest form, it consists of that clonus of the foot and lower leg which appears when the attempt is made to bring the foot quickly into dorsal flexion by pressure upon the sole; a rhythmic, clonic jerking of the foot begins, which ceases as soon as the foot is let go and brought back into plantar flexion. I have demonstrated that this symptom, described by Brown-Séquard[2] and Charcot, most probably originates in the reflex manner, through irritation of the tendo Achillis.[3] In severer cases a very slight pressure on the sole or the toes suffices to produce the phenomenon; for which reason it seems often to arise spontaneously. Then the spasm extends to other parts; the whole leg falls to trembling convulsively, and the other leg soon does the like. When the evil is at its worst, any irritation whatsoever, originating in the skin or the intestines, and acting reflexly, is followed by a tetanic stiffness, together with convulsive tremor, of one or both legs, lasting several minutes. This severest degree of reflex convulsion, which is hardly found except in entirely palsied, paraplegic limbs, is what Brown-Séquard (loc. cit.), and after him Charcot,[1] have rather unsuitably termed *tonic spinal epilepsy*. This phenomenon is especially observed in cases of compression of the cord, or circumscript affections of its entire transverse dimensions, if the reflex excitability is also much increased by an accompanying state of irritation. The slighter degrees of this phenomenon seem also to occur in sclerosis of the lateral columns,[2] while in severer cases we should always think of an implication of the gray substance.

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[1] Volume XI. p. 368.

[2] Journ. de la Physiol. de l'homme et des anim. I. 1858. p. 472.

[3] Ueber Sehnenreflexe bei Gesunden und Rückenmarkskranken. Arch. f. Psych. u. Nervenkr. V. Heft 3. p. 792.

The so-called *saltatory spasms*, recently described by Bamberger, Guttmann, and Frey, would seem to belong to this category, and to present a specially marked modification of these reflex cramps.

It is evident at a glance that this phenomenon has nothing whatever to do with epilepsy proper. But doubtless there are certain relations between epilepsy and spinal disease, which we must briefly speak of in this place.

Brown-Séquard[3] made the remarkable discovery, and studied it with the greatest thoroughness, that in guinea-pigs and other mammalia epilepsy appears in four or five weeks after unilateral section of the lumbar or dorsal cord; an attack may be produced at any moment by irritating an epileptogenic zone, comprising portions of the region of distribution of the trigeminus and the two or three upper cervical nerves. We need not give all the details of Brown-Séquard's experiments, though they are extremely interesting; they have, at all events, demonstrated a fact which has since received confirmation from other sources, namely, that, after unilateral injury of the cord, a diseased condition develops in a few weeks, which presents an unmistakable likeness to epilepsy. We are, however, still in uncertainty regarding the interior relations and mechanism of these processes. Brown-Séquard found also that section of one sciatic nerve produces, after a few weeks, exactly the same epileptiform accidents as the section of the cord. Finally, Westphal[1] found that simple tapping on the heads of guinea-pigs would give rise to an exactly identical form of epilepsy with epileptogenic zone, etc.; he found, in every instance, small hemorrhages, irregularly distributed, in the medulla oblongata and the upper part of the cervical cord, and very often further down, even in the dorsal cord. Westphal is inclined to regard the hemorrhages in the cord as furnishing the cause for the subsequent development of epilepsy.

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[1] Clinical Lectures on Diseases of the Nervous System. 1872–3. p. 216.

[2] *Erb*, Ueber einen wenig bekannten spinalen Symptomencomplex. Berl. klin. Wochenschr. 1875. No. 26.

[3] Compt. rend. de la Soc. de Biolog. 1850. Vol. II.; Arch. de Médic. Févr. 1856; Researches on Epilepsy. Boston, 1856–57; Lectures on the Physiol. and Pathol. of the Central Nervous System. Phila. 1860. p. 178; Arch. de Physiol. norm. et path. I. 1868. p. 317; II. 1869. pp. 211, 422, 496; IV. 1872. p. 116.

It therefore appears to be proved with sufficient certainty that, in animals at least, certain injuries of the cord—as small hemorrhages, and probably other lesions—are capable of giving rise to epilepsy in a manner hitherto unknown. But the question whether such a thing occurs in the human subject is not yet decided with certainty. It is true there are several cases in which epilepsy appeared after lesion of the sciatic nerve, just as in animals; and Leyden[2] has published a case in which epilepsy occurred after an injury of the head, and which seems to resemble Westphal's experiments; but in the case of diseases or injuries of the cord the demonstration of secondary epilepsy has been much less clear. Brown-Séquard quotes some cases from older accounts, which he believes establish this point. Charcot[3] mentions periodic epileptic attacks among the symptoms of compression of the spinal cord, and cites a series of cases in proof of it; a case by Duménil[1] seems especially convincing; and Oppler[2] has quite lately published the case of a young, powerful soldier who had never suffered from epileptic spasms, but had several attacks during the convalescence from a traumatic spinal meningitis. In spite of this, it would be desirable to have more observations upon the human subject.

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[1] Ueber künstl. Erregung von Epilepsie bei Meerschweinchen. Berl. klin. Wochenschr. 1871. No. 38.

[2] Virchow's Archiv. Bd. 55.

[3] Leçons sur les malad. du syst. nerv. II. Sér. 2. fasc. p. 137.

Considering the very great frequency of lesions of the cord, the occurrence of epilepsy as caused by such lesions is extremely rare, and therefore of little practical consequence. It would be of great interest, if cases should be observed, to demonstrate the existence of an epileptogenous zone.

A less common series of spinal symptoms is furnished by the *associated movements*, by which we mean involuntary, often spasmodic actions, which, making their appearance when any voluntary movements are to be executed, complicate and disturb them. The region of their origin seems to be usually the brain, and especially the centres of co-ordination in the brain. No doubt many symptoms in diseases of the cord belong to this class; thus we cannot altogether refuse to regard the atactic movements, the shaking in local sclerosis, etc., as associated movements, although they are decidedly of spinal origin. In the same class are the movements of antagonists, which accompany the effort to innervate palsied or paretic muscles; these, however, are not a true spinal symptom, but simply express the fact that a large number of muscles receive a common co-ordinated innervation, some of which have become insufficient to perform their functions, thereby permitting the action of their antagonists to appear more distinctly; this occurs in all palsies. It is not certain to our mind whether the spasmodic and wholly uncontrolled movements which often occur in paraplegic extremities when a powerful act of the will is directed to them, are to be considered as belonging to this class. They seem to depend rather on an abnormal diffusion of the process of excitation among the motor paths, when pathologically irritated—a diffusion which might be explained by reference to the structure of the nervous network in the inflamed gray substance, and by supposing an interruption of the chief paths to have taken place.

Probably, however, a part is owing to the presence of reflex actions, the stimulus to which originates in the skin of the palsied parts, under the influence of the motions of the trunk and arms; this symptom is almost always associated with an excessive increase of reflex excitability. It is quite certain that the reflex process is concerned in those tonic or clonic contractions of paraplegic legs, which are often associated with the act of evacuating the urine or fæces. The same have been observed by Freusberg in dogs whose spinal cord had been cut.

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[1] Gaz. des hôpit. 1862. p. 470.

[2] Rückenmarksepilepsie? Archiv für Psychiatrie u. Nervenkrankheiten. Bd. IV. p. 784.

It is hardly necessary to repeat, that in all the above symptoms of motor irritation our first thought should be of an affection of the gray substance and the antero-lateral columns. We are at present unable, or at best are able in but a few cases, to define the point where the implication of either terminates, or the general pathogenesis of the spasms. It should not be forgotten that an affection of the sensory portions of the cord may give rise to reflex symptoms of spasm.

e. Alterations in the Velocity of the Motor Conduction.

Recent observations have made us aware that this class of alterations is by no means infrequent; but at present they have been little observed. *Retardation of the motor conduction*, long known by physiologists, was first observed under pathological conditions by Leyden and von Wittich,[1] who made careful estimates of its extent. Their three cases, however, are probably not affections of the spinal cord proper, but of the pons and medulla oblongata. The rate of conduction was reduced to one-third of the normal speed, and in correspondence with this, the movements of locomotion, speech, and so forth, were greatly retarded, and the patients were unable to execute the same movement several times in rapid succession.

The principal attention was directed to measuring the motor conduction as a whole. Burckhardt[2] has lately undertaken to measure the spinal motor conduction alone, by the aid of a variety of physiological methods, and has reached very remarkable results. He found that under normal circumstances *the spinal motor conduction takes place from two to three times as slowly as the peripheral*, and he suspects that the reason lies in the intercalation of ganglion cells in the motor conductive path. But under pathological conditions he found sometimes an acceleration of spinal conduction (as in writers' cramp, in spinal palsy of children, in central myelitis, in some cases of tabes, etc.), and sometimes a more or less considerable *retardation* (as in myelitis of the white substance, in diffuse sclerosis of the cord, in certain cases of tabes, etc.). He concludes that the anatomical basis of central retardation probably lies in the white substance, and that of central acceleration in the gray. Retardation of motor conduction in the cord gives ground, therefore, for inferring a disease of the white substance, and acceleration for inferring disease of the gray.

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[1] Virch. Arch. Bd. 46. p. 476, and Bd. 55. p. 1.

[2] Die physiol. Diagnostik der Nervenkrankheiten. Leipzig. 1875.

It scarcely need be said that these theories are greatly in need of more exact establishment and elaboration.

f. Electric Reaction of the Motor Apparatus.

The labors of Marshall Hall, Todd, Duchenne, and others gave rise to great expectations of improvement in the diagnosis of spinal diseases through the use of electricity; but these expectations have not been fulfilled.

In fact, the electrical examination seldom gives decisive evidence, to enable us to place the seat of a disease in the cord, or brain, or peripheral nerves; this can be done only under special conditions. In many cases, however, valuable information is given as to the state of nutrition of the nerves and muscles, and thereby indirectly as to the nature and probable location of the disease. (Compare the full statements of this matter made in Vol. XI., p. 423 et seq.)

A practically useful general statement of the changes in electrical excitability which occur in diseases of the cord cannot be given at present; the investigations hitherto made are few, and not free from objections. The most frequent, and probably the most important, forms of alteration—namely, the *slight* quantitative changes in electrical excitability—suffer from a serious want of observations; the results have in almost all cases been rendered untrustworthy by defective methods of investigation. I have shown[1] the method which should be observed in seeking

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[1] *Erb*, Zur Lehre von der Tetanie, nebst Bemerkungen über die Prüfung der elektr. Erregbarkeit motorischer Nerven. Arch. f. Psych. und Nervenkr. IV. p. 271.

(particularly in spinal diseases) for certain and exact results. I give here a few points briefly.

In diseases of the spinal cord the electrical reaction of the nerves and muscles, under the faradic and galvanic currents, may be *increased* or *diminished;* the degree of alteration is usually slight. The diminution may amount to a complete extinction of electrical excitability, but this is usually preceded by the change denominated the reaction of degeneration (Entartungsreaction). Distinct pathological conclusions cannot be drawn at present from the slighter degrees of quantitative change.

The *reaction of degeneration*[1] is by no means rare; it is more frequent than has been supposed. It is not yet known whether it takes the same strongly-marked course as in traumatic lesions of peripheral nerves; from certain observations, which, however, are not conclusive, it seems to me that the increase in the susceptibility to galvanic currents is not quite so marked as in peripheral palsies, or perhaps that it passes off more quickly. It is, however, certain that the qualitative change of galvanic excitability occurs in a perfectly characteristic form, with a contraction at anodic-closure which is stronger than that at cathodic-closure (AnSZ > KaSZ), and with a sluggish and protracted contraction.

The inferences we are permitted to make in respect to the histological changes in nerves and muscles, which exhibit such reactions, are the same that may be made in peripheral palsies. They are of a good deal of importance, and the changes they indicate are of the most striking character. By patient care a great number of facts have been collected, of which mention will be made further on, and in the special portion of this work (see "Trophic Disturbances" and the chapter on "Spinal Paralysis of Children"); we have learnt that identical histological changes may originate from direct lesions of the gray substance of the anterior cornua (spinal origin), and from an impediment to conduction between that gray substance and the peripheral parts (peripheral origin). When, therefore, in a disease which can be demonstrated to be of spinal origin, we find the reaction of degeneration, we may infer a lesion of certain parts (anterior cornua) of the gray matter, provided, however, that a peripheral origin can be disproved. On the other hand, when the electrical excitability is retained in its normal form in a spinal disease, it is allowable to infer that these portions of the anterior gray substance are not involved in the lesion.[1]

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[1] See Volume XI. p. 427.

As a very general rule, it may be stated that the results of electrical examination cannot safely be applied to the diagnosis of spinal disease, except in the closest connection with the other symptoms.

We shall give a more detailed presentation of the known facts in connection with the respective diseases.

3. *Disturbances of Reflex Activity.*

It is of the greatest importance to test the reflex activity in spinal diseases; the most valuable information in respect to the disease may often be thus had.

Diminution or suspension of the reflex actions is usually recognized with ease; it is shown by the partial or complete failure of the ordinary means of stimulation, as applied to the skin or to the tendons and other parts from which we expect reflex results. It is necessary to remember that many persons have normally a very slight reflex reaction; but this will be easily recognized, as a pathological diminution is usually limited to a portion of the body.

It may be produced—

a. By *disease* (arrest of conduction) *of the entering sensory root-fibres;* in which case there must be more or less anæsthesia in the regions supplied by these root-fibres.

b. By *disease* (arrest of conduction) *of the departing motor root-fibres;* in which case there must be a more or less complete paralysis of corresponding extent.

c. By *disease of the gray substance* of the reflex apparatus itself, when both sensibility and motility may be present, or one or both of them may be altered to a greater or less extent, according to the extension of the disease in the gray substance. Examples of all these forms are furnished by pathology.

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[1] Compare the observations of *Burckhardt* in Physiolog. Diagnostik der Nervenkrankheiten. pp. 264 and 270.

d. Finally, it is permissible to entertain the case of *reflex arrest,* in accordance with known physiological facts (see above, p. 51); but we have at present no pathological facts.

The decision between these various possibilities will be facilitated by an exact estimation of the weight of each element in the problem.

A *retardation* of reflex actions has been found in pathological cases under the same circumstances as retardation of sensory conduction. We have mentioned this fact previously (p. 70).

A more frequent and interesting circumstance is the *increase of reflex actions.*

It makes its first and its most conspicuous appearance in the *voluntary muscles.* They twitch at the lightest irritation; often the movements are slight and imperfect, but more often they are very powerful, complete, or violent; the legs and arms are thrown about forcibly in all directions, twitch frequently, or enter into a clonic tremor or a fully developed tetanus; the motions are almost always unsuitable and disorderly; flexion and extension of the joints alternate, giving rise to irregular sprawling movements; the patient's leg can often be placed at will in reflex extension or flexion by a definite localization and intensity of irritation;[1] orderly and fitting movements are rarer; thus McDonnel[2] reports that a patient, who suffered with compression of the cervical cord, continually moved his paralyzed left hand to his genitals during catheterization.

Such reflex acts are usually produced with most ease by irritating the *skin;* tickling, pricking and pinching, or stroking the skin, especially that of the soles, the inner surface of the thighs, the toes and fingers, and the palm, produce them; Pflueger's law of extension of reflex phenomena can often be confirmed in such patients. Reflex acts are also excited from the intestines; in certain spinal affections nothing is more common than to find full, powerful, and very troublesome reflex actions taking place in the legs during evacuation of fæces or urine, in consequence of colic pains, or during catheterization; they often continue for a considerable time in a semi-rhythmic manner. The reflex actions which originate in tendons, fasciæ, and articular ligaments are also very important. We have given above (see p. 48 et seq.) a brief account of their physiological occurrence; in pathological cases they often increase to such a degree that the lightest tapping on the tendons leads to the most vigorous jerking; in this case the phenomenon can be produced from many more tendons than usual; thus, I have seen them originate from the ligamentum patellæ, the tendo Achillis, the tendons of the adductors, gracilis, biceps femoris, tibialis anticus and posticus; in the arm, from those of the triceps, supinator longus, radial extensors, flexors of the fingers, biceps, flexor radialis, etc. Their relation to the reflex actions of the skin is very variable; sometimes both are present and are exaggerated, sometimes those of the tendons are absent while those of the skin are present, and sometimes those of the tendons are enormously increased while those of the skin are normal or diminished. These circumstances are probably of great importance in a diagnostic point of view, but this requires further observation.

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[1] *Virchow,* Gesammte Abhandl. p. 683.

[2] See Virchow-Hirsch's Jahresber. 1871. Bd. II. p. 7.

The class of tendon-reflexions ought, in our opinion, to include a reflex phenomenon which has long ago been described by French authors (Brown-Séquard, Charcot, Vulpian, Dubois, and others)—namely, the *reflex clonus* which appears in the foot and lower leg *when the foot is rapidly brought into dorsal flexion by another person.* If the fore-part of the sole is vigorously pressed up with the flat of the hand, the rhythmic reflex contractions of the calf produce a clonic tremor of the foot, which continues as long as the pressure on the sole lasts, and ceases immediately when the pressure is withdrawn or when the foot is brought energetically into plantar flexion. When the reflex excitability is greatly increased, the slightest pressure on the sole is sufficient to set in motion the clonus; the action may extend to the whole leg, and even to the other leg; in its most violent forms, tetanic rigidity of the legs alternates with convulsive shaking, and we have the phenomenon described by Brown-

Séquard and Charcot as *spinal epilepsy* (see p. 101). In my article on Tendinous Reflex Actions[1] I have attempted to prove that this reflex clonus is nothing but a tendinous reflexion, produced by the sudden tension of the tendo Achillis, and maintained in a very simple way by the continued pressure against the foot. I have recently succeeded more than once in producing just the same phenomenon from the tendo patellæ, and have observed it in the biceps femoris. It also occurs in the foot, when the skin is irritated, if the excitability is very much exalted; I have explained (p. 50) the mechanism of this. It cannot be denied that the phenomenon may possibly be produced directly from the skin in many cases; but we require more convincing proof than Joffroy[2] has brought forward.

The increase in reflex activity may manifest itself in the *intestines* and the *vaso-motor apparatus;* but this has been little studied in the human subject. For example, I have observed in paraplegic patients that a sudden discharge of urine can be produced by external pressure upon the bladder, or by introducing the finger into the rectum; I have also seen a mucous fluid evacuation of the bowels occur regularly upon the occasion of dressing and cleansing a large bed-sore; I also have seen erections produced by irritation of the skin of the penis or perineum, or by introduction of a catheter, etc. It is probable that a closer inspection will frequently discover a reflex action upon the cutaneous vessels, etc.

This excess of reflex action is explained, first by a *separation of the reflex apparatus from the brain*, which puts a stop to the action of the centres of inhibition. In fact, the highest degree of excess is found in all those affections of the spinal cord which put a complete stop to the transmission of impressions in the cord; in separation or compression, in circumscribed transverse myelitis or softening, in tumors or cavities of the cord, etc. It must be considered an indispensable condition for the production of the reflexions, that the gray substance situated below the point of lesion should be intact; and it is not strange that parts which have retained their reflex excitability usually possess also their electrical excitability, because the gray substance is the essential factor in the conservation of the nutrition of nerves and muscles.

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[1] Arch. f. Psych. u. Nervenkr. Bd. V. p. 792.

[2] Gaz. méd. de Paris. 1875. Nos. 33, 35.

The second cause of increased reflex action is the *increased excitability* of the *gray substance*—a condition which may certainly be produced by inflammatory and other irritative conditions, by many sorts of disturbance of nutrition, and by certain poisons, as strychnia, opium, belladonna, etc. The greatest increase will be found when both these causes act together, as in the case of myelitis from compression, or when strychnia is acting on the paraplegic parts; this is confirmed by daily experience.

Whether an increase of the excitability of the sensory conductors (hyperæsthesia), or of the motor conductors (convulsibility) is capable by itself of producing an increase of reflex activity, is a point which requires closer examination, but is not improbable *a priori*.

4. *Vaso-motor Disturbances.*

These are very frequent. They consist of the two conditions of spasm and palsy of the vessels, with the consequences. In view of the complicated mechanism for innervation possessed by the vessels, explained on page 42, it is extremely difficult to interpret these phenomena, especially as there are many others to be taken into consideration along with them.

The following appear to constitute the principal facts:

In many cases *local hyperæmia and fluxion*, *increased redness*, and *elevation of temperature* occur in the parts affected (usually paralyzed); thus, for example, in complete severance or compression of the cord, of acute origin; and most distinctly of all, in unilateral lesions, where the difference between the diseased and the well side is very striking. If the lesion is entirely local, and the vaso-motor centres situated within the cord are left nearly intact, the normal condition returns after a short time. In such cases the symptoms of vascular paralysis are mostly temporary, and the normal condition returns after some weeks or months; while at a later time paleness, diminution of temperature with subjective feeling of cold, and even cyanosis, are very often observed.

In other cases, especially in the early stages of tabes or myelitis, before any symptoms of palsy appear, there is a *striking coldness, great paleness, and anæmia* of the lower extremities, a tendency to goose-skin, the subjective feeling of icy coldness of the feet and inability to warm them even in bed, a small pulse, excessive sensitiveness to the impressions of cold, etc. It cannot be doubted that these phenomena are due to conditions of abnormal excitability of the vascular nerves, with increased contraction and excitability of the vessels.

Finally, in very severe or old cases (usually of spinal palsy) we observe, in connection with the subjective and objective coldness of the parts, a *marked lividity, a cyanotic tinge of the skin,* puffiness, swollen veins and capillaries, slow and poor circulation; in this case there evidently exists a state of great vascular atony, and a more or less distinct venous stagnation; the arterial pressure is lessened, the venous increased, the circulation retarded.

These three groups of vaso-motor disturbances may be naturally interpreted by what we know of the laws of vascular innervation.

In the first group—paralytic hyperæmia—we should recall the hundreds of experiments that have been made upon section of the cord; every such section produces paralytic hyperæmia in the parts situated behind it. Goltz has shown how, when the spinal centres recover themselves, the circulation may return to nearly its normal point; only when the destruction of these centres is quite extensive (as in spinal apoplexy, hæmatomyelitis, etc.) will the fluxion and elevation of temperature be more permanent, and finally result in atony of the vessels.

For the second group—ischæmic pallor and coldness—abnormal excitement of the vaso-motor centres and conductors is doubtless responsible; they are most frequently caused by chronic inflammatory conditions of the cord, and may be produced either directly or reflexly.

In the third group—atonic hyperæmia by stagnation—it is commonly supposed that the absence of muscular action in the palsied limbs produces disturbances of the venous circulation, and thence gives rise to the other symptoms. It may, however, be easily seen that this cyanosis occurs in parts which are not at all palsied, and may be absent in completely palsied regions. The assigned explanation, therefore, is at least insufficient; the absence of muscular contraction may favor the production of atonic hyperæmia, but cannot by itself produce it. It seems to us that, in order to produce such results, a more or less general palsy of the vaso-motor nerves is required, such as is produced either by destruction of the vaso-motor centres in the gray substance, or by interruption of the vaso-motor conduction in the lateral columns or the anterior roots. The longer this paralysis exists, and the more complete it becomes, the more marked will be the vascular atony. Spinal palsy of children (destruction of the vaso-motor centres in the gray substance) is a good example of the former case; in disease of the lateral columns the question has not been fully studied, but in one case of probable sclerosis of the lateral columns I have observed an exquisite development of the atonic hyperæmia of stagnation.

Conditions of great vascular irritability in the skin are occasionally observed; sudden blushing or paling of certain portions of skin, erythema fugax, etc. They appear most frequent in association with meningitic conditions.

It therefore appears plainly that vaso-motor disturbances may afford us some information respecting the nature of the disturbances in the cord, but enable us to draw no certain conclusions respecting its exact locality.

5. *Trophic Disturbances.*

These constitute one of the most interesting, and also one of the obscurest symptoms of spinal disease, and there is still a great deal of controversy in regard to their interpretation. We must necessarily speak of the different tissues separately, in our enumeration of these symptoms.

The trophic disturbances of *nerves* and *muscles* have received great attention, especially in spinal diseases; it has very lately been discovered that they may occur apart from palsy, becoming a subsequent cause of palsy, as in the typical form of progressive muscular atrophy.

But it is not all the forms of spinal paralysis that are accompanied by considerable trophic disturbances of nerves and muscles. In many cases the nutrition and the electrical excitability of these parts suffer not the slightest change; such cases are always due to circumscribed disease of the entire transverse section of the cord, or a disease of the white substance, which may be of great extent. The affection of these parts seems, therefore, to have nothing to do with the nutrition of the nerves and muscles; an inference which is certainly correct in regard to the entire posterior columns and the posterior section of the lateral columns, but is still doubtful in respect to the portions of the anterior columns which are traversed by the anterior roots. It seems certain, however, that in all cases of paraplegia without atrophy the gray substance—at least that of the anterior columns—is not altered to any considerable extent.

There are also cases in which a *simple atrophy of the muscles* occurs, which may make great progress, and may even emaciate the legs to the condition of a skeleton; the histological condition of the muscles remaining for the most part intact, with only a diminution of the breadth of the fibres, and in places a certain increase of interstitial fatty tissue, but without a trace of proliferation of interstitial connective tissue, of multiplication of the nuclei in the muscles, etc. In correspondence with this condition, the electrical excitability remains entirely intact, with, at the most, a slight quantitative diminution, and the reflex actions are mostly present.

This form of atrophy occurs in the later stages of tabes, in many cases of chronic myelitis, in paralyses due to compression from vertebral caries, etc.; it appears most frequently to attack persons who are debilitated or weakly. This simple atrophy is commonly referred to the palsy, the long-continued disuse of the parts, but we are not content with this explanation in all cases. We cannot but think that certain distinct changes in the spinal cord must exist before this form of atrophy can come to pass. At all events, this question is still in great need of investigation, both in its histological and its pathogenetic relations. Our present knowledge of it is extremely small.

The same is true to a certain point of the most important form of disease of nutrition in nerve and muscle, the *degenerative atrophy*. In certain spinal affections, this is developed very quickly and in a strongly-marked form. Although the histological account of this disease is in many respects defective, especially in the earlier stages, yet our present knowledge, in connection with the results of electrical examination (which invariably gives the "reaction of degeneration") points with tolerable certainty to the conclusion that the essential histological changes are the same as those which so invariably follow severe traumatic lesions of peripheral nerves—fatty degeneration and atrophy of the nerve-fibres, simultaneously with proliferation of cells and hyperplasia in the neurilemma; atrophy with increase of nuclei and chemical changes in the muscular fibres, simultaneously with proliferation of the interstitial connective tissue; and in the last stages excessive loss of muscular substance and transformation into connective tissue with secondary deposition of fat. We would refer the reader to the full description of degenerative atrophy of the nerves and muscles given in Volume XI., p. 411. We are convinced that the changes are essentially the same; it remains, however, to be decided by more careful examination whether the process in spinal diseases runs its course with the same rapidity and intensity as in traumatic palsies, and whether there are not certain differences in degree, which require further statement.

This degenerative atrophy occurs regularly in the so-called spinal palsy of children and the analogous affection of adults; it is regularly found in the typical form of progressive muscular atrophy (atrophie muscul. progress. protopathique of Charcot); it appears to occur also in the sclérose latérale amyotrophique described by Charcot,[1] and in many other spinal affections, whenever they implicate the gray substance of the anterior cornua.

The past few years have given us a series of discoveries in relation to the spinal palsy of children and progressive muscular atrophy (which we may regard as types of the spinal affections here considered), which indicate a great improvement in our knowledge of the trophic functions of the spinal cord. It has been shown that these diseases are uniformly associated with an affection of the anterior cornua, in the former case acute, in the latter chronic, which regularly and in quite a peculiar way involves the great motor ganglion cells. The most recent observations, without exception, state this point, which was formerly almost always overlooked, owing to defective methods of examination. It would lead us too far, if we were to undertake to enumerate and criticise all these observations. We would refer to the memoirs, chiefly published in the Archives de physiol. norm. et pathol., by Charcot, Joffroy, Hayem, Duchenne, Vulpian, Pierret, Gombault, Troisier, and others, and the experiments of Voisin and Hanot, Lockhart Clarke, Roger and Damaschino, Roth, and others, which are thoroughly convincing. These observations contain in addition a series of facts which decidedly refute the view that the affection is due to an inflammation conducted from the primary focus in the cord to the nerves and muscles; but further special observations on this point are desirable. Observers are not agreed as to whether the change of the ganglion cells is primary, or whether it is the consequence of an interstitial myelitis. That, however, is a subordinate question, as concerning the object of our study. It appears to be certain, at any rate, that functional injury or destruction of these great ganglion cells of the anterior columns is most closely related to degenerative atrophy of the nerves and muscles.

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[1] Leçons sur les maladies du système nerveux. II. Sér. 3. fascic. p. 213 et seq. 1874.

It certainly can hardly be doubted any longer that the trophic centres for the motor nerves and muscles are situated within the gray substance of the cord, very close to the point where the anterior roots of the nerves concerned enter. While this is rendered exceedingly probable by the above facts of local destruction of the anterior cornua, it is on the other hand strongly supported by the fact that the degenerative atrophy is absent, even in cases of the severest spinal paraplegia, when the corresponding sections of the gray substance are intact. Compare upon this point two cases given by Burckhardt.[1]

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[1] Physiol. Diagnostik der Nervenkrankh. p. 264. Beob. 45 und 46.

Of the nature of the connection between these trophic centres and the peripheral parts, of the manner in which, and the channels through which their influence is distributed from the one to the other, we are ignorant; and the boldest hypotheses are allowable. Anatomy and physiology are not informed of the existence of proper trophic nerve-paths; but of those who write upon the subject, one party regards them as a physiological postulate, while the other refers the transmission of trophic influences to the motor and sensory fibres. I have attempted to show,[1] by the comparison of a series of mutually corroborative cases, that the trophic paths cannot be fully identical with the motor. Those facts certainly show that the motor and the trophic paths must be distinct at some points, as they are capable of being diseased independently of each other. Such separation certainly exists in the central organ; but it is as yet doubtful how far outwards it extends—whether motor and trophic fibres run separately as far as the periphery, or whether the motor fibres are also capable of conducting the excitation from the trophic centres. Burckhardt[2] is of the latter opinion, and claims for the great ganglion cells of the anterior cornua the function of centres of nutrition for the motor fibres which pass from them, and for the muscles. In fact, the recent investigations into the structure of the ganglion cells furnish a ready and plausible form for conceiving of the double or manifold functions of these great ganglion cells. If the plan which Max Schultze[3] gives of the fibrillary structure of the ganglion cells is correct in the case of the human subject —and that can hardly be doubted—it can easily be conceived how such a cell may form the point of union of fibrils of the greatest variety of physiological rank (motor, co-ordinatory, reflectory, etc.), which unite, in part, in the nerve-process, and enter the anterior roots. While thus the cell serves as the point of union for these various fibres, it is also capable of acting as a trophic centre for them, and of sending from its own substance trophic fibrils to the anterior root-fibres. The well-known fibrillary composition of the axis-cylinder would even permit us to dispense with the hypothesis of trophic nerve-fibres, as the axis-cylinder of the motor nerve may easily be supposed to contain fibrils of very various physiological functions. We will not, however, enter too far into the field of purely hypothetical considerations.

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[1] Ein Fall von Bleilähmung. Arch. f. Psych. u. Nervenkrankh. Bd. V. 1875. p. 445.

[2] Loc. cit. p. 271.

[3] Stricker's Handb. der Gewebelehre. p. 130.

One last question is still awaiting a decision—the question whether the processes of degenerative atrophy should be referred to an *irritation* or a *palsy* of the trophic central apparatus. Charcot[1] is of the former opinion, thinking that irritation of the trophic ganglion cells and the trophic fibres produces degenerative atrophy, while simple paralysis, or separation of the cells from the peripheral parts, leaves the nutrition of the latter intact. He bases his opinion chiefly upon the supposed fact that crushing, inflammation, and similar injuries of the peripheral nerves act in a different way from simple section; in the latter case the characteristic histological changes being supposed not to occur. The incorrectness of this position is sufficiently proved.[2] The processes of infantile spinal paralysis also render this assumption quite impossible; for, firstly, the initial phenomena, the complete palsy, etc., make an increase of the activity of the ganglion cells very improbable; and secondly, the complete disappearance of the cells, which is demonstrated in the later stages of all such cases, would necessarily (upon Charcot's theory) result in repair of the disturbances of nutrition, so that the nerves and muscles would return to their normal state — which is the contrary of what we know to be the case. We are, therefore, at present of the opinion that *a paralysis or destruction of the central trophic apparatus, or a separation from the peripheral parts, produces the symptoms of degenerative atrophy.* This may make the matter harder to understand, but we are obliged to take the facts as they are.

Upon the whole, we are justified by the present state of our knowledge in assuming a disease of the anterior cornua when the electrical examination shows the existence of the reaction of degeneration, and consequently of degenerative atrophy of nerves and muscles, provided the disease is clearly of spinal origin.

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[1] Leçons sur les maladies du système nerveux. 1872–3. pp. 19, 23, and ch. 2.

[2] *Erb*, Zur Pathol. und pathol. Anat. peripherer Paralyse. Deutsch. Arch. f. klin. Med. Bd. V. p. 53.

It is well known that an increased *deposit of fat* in the interstitial connective tissue of the atrophied muscles is not rare in the later stages. I have very recently seen a case of palsy from spinal apoplexy, in which the muscles of the calf, at first atrophied, became by degrees excessive in size, probably from deposition of fat, the paralysis continuing.[1] This is a kind of *pseudo-hypertrophy* of the muscle. But it is still a controverted point whether the peculiar disease known by this name (also atrophia musculorum lipomatosa, paralysie musculaire pseudohypertrophique, etc.) is of spinal origin; Charcot, Eulenburg, and Cohnheim are opposed to this view; L. Clarke, O. Barth, and others, are in favor of it; Friedreich considers the disease as a mere progressive muscular atrophy modified by certain peculiarities of infancy; and W. Mueller likewise considers the lipomatosis as a more or less accidental complication of the atrophy. At all events, we must wait for further investigation of the matter.

The same must be said of those cases of *true muscular hypertrophy*—rare at present—which have been found sometimes alone (Auerbach, Berger), and sometimes accompanied by progressive muscular atrophy (Friedreich[2]). The neurotic origin of these cases cannot at present be established. They should not be confounded with the hypertrophy from use, which occurs here and there in muscles which have had to do double duty on behalf of paralyzed ones. A good instance of this is presented by the left sartorius, in the case described by me, as above mentioned.[3]

Trophic disturbances in the *structures of the skin* are very common in spinal disorders. Those of the epidermoid structures are of subordinate importance, although their theoretic interest is considerable. For instance, in some cases of spinal palsy an abnormal growth of *hair*[4] has been observed. Closely associated with other disturbances of the skin, with atrophy of the muscles, etc., occur decided alterations of the *nails*, deformity, increased curvature, and furrowing, clubbed swelling, yellowish or brownish discoloration, and the like. The changes of the *skin* itself are more important.[5] First, and not infrequently, *erythematous*

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[1] Arch. f. Psych. u. Nervenkrankh. Bd. V. Heft 3. p. 782.

[2] Ueber progress. Muskelatrophie, ueber wahre und falsche Muskelhypertrophie. Berlin. 1873. Cap. VI.

[3] Loc. cit. Bd. V. p. 780.

[4] *Jelly*, Brit. Med. Jour. 1873. June 14.

[5] Compare in particular the beautiful account given by Charcot in his Clinical Lectures.

spots and eruptions, which may appear almost anywhere, and usually are very transient; then *lichenoid* or *papulous* eruptions, extending over greater or smaller portions of the skin, often restricted to the district of a single nerve or to a single limb; not infrequently wheals of *urticaria*, of greater or less size and extent, usually accompanied by violent itching; or *herpes zoster*, in its characteristic mode of appearance; and finally, in rare cases, *pustule-formation*, related to pemphigus and ecthyma, and usually leading to ill-looking and badly-healing ulcerations.

Besides these alterations, which remind us of the appearances of well-known and typical diseases of the skin, there occur others of a more diffuse sort; uniform thinning of the skin, abnormal smoothness and shining look of the epidermis, connected with more or less livid redness ("glossy skin")—changes of which we have spoken in Vol. XI., p. 409, under Paralyses; on the other hand, there are often abnormal thickening and swelling of the skin and subcutaneous cellular tissue, not seldom connected with œdema, or gradually developed from it.

Our knowledge of the theory of all these phenomena and their special connection with the spinal cord and with the diseases of certain sections of it, is still very defective. The idea that they may be of neurotic origin is hardly yet familiar, but from what parts they may originate, and how, is still a matter for guesses merely. In some cases they are usually associated with violent sensory phenomena (the lancinating pains of tabes, etc.); hence it has been inferred that they might depend on an irritation of trophic fibres—a position which is strongly supported by the manner in which herpes zoster occurs in neuralgia and after neuritis; on the other hand, it is not to be denied that another group of these phenomena occurs only under circumstances which almost conclusively point to paralysis of the trophic paths. At present it cannot be decided with certainty what special part the irritation or paralysis of trophic fibres takes in the production of cutaneous changes.

The peripheral channels through which the trophic influence passes to the skin and its adnexa appear beyond a doubt to lie in the sensitive nerves; this is supported by a large number of unquestionable facts. But it is still doubtful where the centres for the nutrition of the cutaneous structures are situated. They appear, at all events, not to be in the anterior cornua; this follows from our experience in regard to the spinal paralysis of children. And, as the gray substance is the only probable region, there remain only the posterior cornua, or the spinal ganglia; the latter site has been rendered probable by many facts, and is accepted by several authors. We must leave it for the future to establish the influence of these structures upon the nutrition of the skin.

By far the most important trophic disturbance which the skin experiences in spinal disease is *gangrene from pressure* or *bed-sore.* Its occurrence is a sign of the greatest danger, and is often decisive of the prognosis. In practice it is well to distinguish two forms: the one is acute, is caused by slight irritation or brief pressure, is preceded by an inflammatory eruption, appears a few days after the beginning of the central lesion and leads rapidly to gangrenous destruction (decubitus acutus of Samuel); the other, more chronic in its origin, accompanies the later course of spinal diseases, is chiefly dependent on prolonged pressure, and takes the form of simple gangrenous death of the skin and the subcutaneous tissue (decubitus chronicus).

Decubitus acutus, of which Charcot (l. c.) has given a very vivid picture, usually begins a few days after any severe spinal lesion or after a severe exacerbation of a spinal disease, and is characterized by a very rapid development. Upon a portion of skin which has been exposed to pressure or to any irritation (and often when no such circumstance has occurred), erythematous spots appear, which are soon covered with vesicles, the contents of which, at first clear, become rapidly brownish or reddish; under favorable circumstances these vesicles may dry up, and the spot recovers without further change; but this is not commonly the case; the vesicles burst and leave ill-looking ulcerations, the basis of which is composed of the skin, infiltrated with blood, and usually in a state of phlegmonous inflammation. The base of the ulcer perishes by gangrene, the neighboring skin is infiltrated with blood, and inflamed to a greater and greater extent, the gangrenous destruction goes deeper and deeper, laying bare and including in its destructive operations tendons, fasciæ, ligaments, and bones.

This entire cycle of processes is complete in a few days, and cannot be avoided by any care or cleanliness; at the same time, cystitis and hæmaturia are sometimes observed, or the muscles become the prey of a rapid atrophy.

The consequences of such extensive gangrene soon appear; violent fever with severe chills and great variations of temperature, a septicæmic condition, purulent infection, gangrenous thrombosis and embolism occur, and the fatal result is preceded by a general marasmus; or the gangrene extends still further into the interior of the vertebral canal, when a purulent or ichorous meningitis, rapidly ascending to the cranium, soon closes the painful scene.

Simple chronic decubitus usually originates in a somewhat different way. In chronic diseases of the spinal cord, in paraplegias, there is seen a diffuse dark redness of the portions which are most pressed upon in sitting or lying; often mingled with superficial ulcerations. Some day there appears upon this reddened portion of skin a black spot, which rapidly enlarges if the pressure is continued. The skin dries to a black leathery mass, around which a bounding line of inflammation soon appears, which under proper treatment leads to a throwing off of the gangrenous layer, and cleaning up of its base, with the formation of granulations. But if the pressure is continued, the ulcerated surface assumes a bad, unwholesome color from infiltration with blood, the inflammation assumes an appearance more like that of a phlegmon, and the gangrene may spread rapidly, deeply, and cause horrible destruction. And then the symptoms above named as characteristic of acute decubitus appear and soon put an end to the patient's life.

This form of gangrene may occur at any place which is exposed to a continued pressure, but is far the most common on the coccyx and buttocks, next over the trochanters and ischiatic protuberances, heels, and knees, the spinous processes of the vertebræ, the shoulder-blades, elbows, etc. In severe cases of this sort, the patient, with his numerous large sores, presents the very picture of wretchedness. Nor is the end of this misery always speedy; if attention and care be given, the sores often clean up, good granulations appear; but the tendency to heal is very slight, and it is a desperate while before cicatrization occurs. I have seen a patient, who had nine large bed-sores and several small ones, linger along for over a year under faithful care. Though some places may cicatrize, a new sore will appear here or there; the occurrence of bed-sores, therefore, unless they improve rapidly and considerably, must always be considered a very bad omen for the patient. The occurrence of acute decubitus renders the prognosis extremely bad in all cases.

Very various accounts are given of the way in which bed-sores originate.

Continuous *pressure* is commonly thought to be the chief cause of the bed-sore; its action is supposed to be considerably favored by the complete immobility of paralyzed patients, by the absence of sensation in some others, by the filth from the bladder and rectum which is liable to accumulate; the more of these causes are present, the more confidently is the occurrence of bed-sores expected.

In fact, we may count with certainty on the appearance of bed-sores when all these causes coincide. But this by no means proves that the sores are due to such causes. In acute myelitis of the anterior columns (spinal paralysis of children, etc.), in many forms of hysterical palsy, in fractures of the thigh, and similar cases, we find abundant proof that long-continued pressure, palsy, etc., are not by themselves adequate to produce bed-sores. Charcot has seen decubitus acutus occur, even when all pressure and all befouling of the person was avoided.

It seems therefore absolutely necessary to seek for another explanation. The vaso-motor paralysis, which is commonly present, and the anæsthesia, have been thought of; but it is easy to prove that this view is incorrect, and that both these circumstances act only as facilitating the action of pressure, which itself is not the essential element in the production of bed-sores.

It is manifest that there must be some very special changes in the nervous system, special influences added or subtracted, in order to produce such fearful gangrenous destruction in patients whose general health is good, and the action of their hearts vigorous. Such influences may most properly be called trophic. In chronic decubitus we may assume that the trophic disturbances cause a diminished power of resistance, a lessened vital turgor, on the part of the skin, which, in connection with the weakness of the circulation caused by the vaso-motor paralysis, probably explains the gangrene of pressure; as to acute decubitus, Charcot believes that he has proved that it is caused by a "violent irritation of a more or less extensive district of the spinal cord."

Acute decubitus is found associated chiefly with severe traumatic lesions of the cord (compression and destruction *en masse* by fractures of the vertebræ, etc.), in acute myelitis, hæmatomyelia, and the like; it has also been observed in unilateral traumatic lesions of the cord, upon the anæsthetic side only, not on the paralyzed side. Chronic decubitus is found in chronic myelitis transversa (affecting the cord diametrally), in the last stages of tabes, in paraplegiæ of slow origin, and similarly also in peripheral palsies in the district of the cauda equina. If the latter originate acutely, *e. g.*, by fracture of the sacrum, they may themselves be followed by acute decubitus.

It seems to us that the one element which the above affections share in common is rather the destruction and paralysis of certain parts than their irritation, and we think it most probable that the chief cause of the decubitus in spinal affections is the paralysis of certain trophic centres in the cord, or their severance from the peripheral parts; in certain cases of acute decubitus, however, it is not yet proved that the disease does not originate in irritation.

The exact location of these trophic centres is as little known to us as is the way in which they exercise their trophic influence upon the skin. Many things render it probable that they are to be found in the gray substance, especially in the central portions and the posterior cornua, and that the paths which begin in these parts are situated in the posterior roots. Observations of unilateral lesion of the cord have also made it probable that the trophic fibres for the skin decussate in the cord like the sensory fibres. The relations of the spinal ganglia to these processes are not clear.

The conclusions in regard to the seat and nature of the spinal affection, which are to be drawn from the occurrence of bedsores, are obvious.

The *bones* often undergo trophic disturbances in spinal disease. One of the commonest symptoms in the spinal paralysis of children is *arrest of growth of bones*. The bones of the extremities remain short and small, the limbs are shortened, the pelvis displaced, and the spinal cord in consequence curved. If a series of cases be compared, it will plainly appear that this impairment of growth is not always proportional to the muscular atrophy or the degree of palsy ; it is to a certain extent independent of these circumstances, and in such cases the paralysis and muscular atrophy may be the chief feature in one limb, and the atrophy of the bone in the other.

In other cases, on the contrary, a *swelling*, *thickening*, *hypertrophy of the bones* is observed ; they become heavier, and are often greatly enlarged, especially at the joints. This condition may coexist with pseudo-hypertrophy of the muscles, or with degenerative atrophy. A closer examination, however, shows that in all such cases the essential element is a hyperplasia of connective substance, both in the bone-tissue and in the muscles.

An abnormal *brittleness of the bones* has been observed in very rare cases.

There can be no doubt that the majority of these changes depend on disturbances of the nervous system. The facts of infantile spinal paralysis render it almost certain that the trophic centres for bones are in the anterior cornua, but that they are certainly not identical with those for the muscles. Further exact study of these conditions is needed.

Especial attention has been lately given to the trophic disturbances of the *joints*, which occur in many spinal diseases, and are very interesting.

It is very common, both in spinal and peripheral palsies, to find slight swelling, stiffness, a little pain, and a moderate amount of ankylosis of the joints. This is probably due in part to long disuse, and occurs in like manner after the long wearing of a plaster of-Paris bandage, but it is partly, no doubt, of nervous origin.

Charcot's admirable researches[1] have very recently placed before us an extremely characteristic affection of the joints, of manifestly nervous origin, chiefly associated with tabes; its characteristic symptoms are an abundant serous discharge, wasting of the cartilages and bones, subluxations, and the like. This is the so-called *spinal arthropathy of tabes.*

This affection of the joints is very strikingly different from the ordinary spontaneous inflammations of a rheumatic or traumatic origin. It much prefers the knee-joint; after that, in diminishing frequency, the shoulder, elbow, hip, and wrist joints. It begins, usually suddenly and unexpectedly, without any external cause, and its first symptom is a large diffuse swelling of the joint, caused by an abundant exudation of serous fluid in its cavity; fever, redness, and pain are usually altogether wanting. The adjoining parts are always considerably swollen, which often extends to nearly the entire extremity. This swelling is partly œdematous, and partly of a harder nature. It usually disappears in a few days and the articular effusion likewise, after which the ends of the bones come in contact, are worn away and their cartilages and ligaments destroyed; these changes are indicated by a good deal of cracking and rubbing noise in the joint. Deformities of the joints are the result—subluxations, loose joints, and the like. This may continue for months or years and then disappear, but usually the changes which result are incurable.

These advanced stages, when examined anatomically, exhibit the marks of arthritis sicca, but with this distinction: that the erosion of the articular extremities is considerably greater than the growth of new bone.

This arthropathy is most common in the early stages of tabes, and chiefly in the preliminary period, before the atactic disturbances of motion have appeared, and when the lancinating pains constitute the chief feature of the complaint. It has, however, been observed in the same, or nearly the same, form in compression of the cord from vertebral disease, in acute myelitis, progressive muscular atrophy, traumatic unilateral lesion of the cord (on the palsied side), etc.

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[1] Cf. *Charcot*, Arch. de Physiol. I. 1868; II. 1869; III. 1870 (with *Joffroy*); and Clinical Lectures on Diseases of the Nervous System. I. and II. series.—*Ball*, Gaz. des Hôp. 1868 and 1869.—*Buzzard*, Lancet. 1874. Aug. 22.—*Weir Mitchell*, Amer. Journ. Med. Sci. April. 1875. p. 339.—See also below, section on Tabes Dorsalis.

It can hardly be doubted that this arthropathy depends on disturbances of the nervous system. Charcot has supposed that they were referable to a pathological irritation of central trophic apparatus, and has, in fact, found in autopsies of tabetic patients suffering from this arthropathy, an atrophy of corresponding portions of the gray anterior pillars and disappearance of their ganglion cells. But in a later case he has failed to find this lesion, in spite of careful search, which discovered marked alterations in the spinal ganglia. Further examination is therefore needed to decide this difficult question. The rarity of the arthropathy in spinal palsy of children, and its close connection with tabes, certainly require of us great care in forming an opinion.

Of trophic disturbances of the intestines in spinal disease we know little, and this little will be set down under the proper heads hereafter.

The *general nutrition*, in most spinal diseases, suffers only in very exceptional conditions, or when the duration is very protracted. It is not rare to see a spinal patient in the most hopeless case—paraplegic, or excessively atactic—confined to his bed or the wheeled chair, who nevertheless looks very well in complexion, is muscular and plump, has a good appetite and digestion, and for years and tens of years enjoys a tolerable existence. In other cases the nutrition fails fast, the general health is much impaired, the patient sinks rapidly into marasmus. The causes of this loss of health are as follows: continued rest, want of motion and fresh air, poor digestion, severe pain which takes away sleep, fever, malignant new-formations, and, above all, cystitis and bed-sores. We shall see in the course of this account that these causes are very frequent, and accompany all sorts of spinal disease.

The *state of the general bodily temperature*, and the *fever* of spinal diseases, may here be mentioned. The local changes of temperature, limited to single extremities or parts of bodies, are accounted for by the vaso-motor disturbances

Inflammations of the cord are accompanied by fever, like those of the other organs. Its type and course will be described under the several diseases of the cord, as acute meningitis, acute myelitis, acute spinal paralysis, paralysis ascendens acuta, etc. Fever also occurs in consequence of many complications of spinal disease, as in gangrenous bed-sore, etc. This is of subordinate importance at present.

On the other hand, the often enormous rise of temperature which accompanies or closes many severe spinal diseases, and which is of great theoretic interest, deserves a brief mention here. This phenomenon is commonly ascribed to the "neuroparalytic agony," and has been frequently observed and several times critically described in severe disease of the various parts of the nervous system, especially in severe lesions of the cord.[1]

The chief interest for us lies in those cases where, after crushing and injury of the cervical medulla, the bodily temperature continued to rise, and finally reached an enormous height (42.9–44.0° C.=109° to 111.3° Fahr.), followed by death. The first case of this kind was observed by Brodie; other similar ones are given by Billroth, Simon, Quincke, Fischer, and others. Unusually high temperatures have also been observed in the agony of tetanus (Wunderlich), of meningitis cerebrospinalis (Erb), etc. Very lately, J. W. Teale[2] has published a case of spinal disease, perhaps inflammatory, caused by an injury, in which the axillary temperature several times reached the incredible height of over 50° C. (122° F.); the case nevertheless recovered.

With a view to explaining the connection between this rise of temperature and the spinal lesion, various experimental observations have been made which, however, have not yet furnished conclusive results. It has been found that section of the dorsal cord produced a depression, while section of the cervical cord in the neighborhood of the pons produced a considerable rise of temperature (Tscheschichin); that crushing of the cervical cord uniformly raised the temperature, if peripheral cooling was prevented by suitable measures (Naunyn and Quincke); and finally, that an injury of the cervical cord produces no rise of temperature, provided the anterior columns are spared (Fischer).

It would lead us too far, to attempt to give the conclusions drawn from these experiments in respect to the exciting and moderating influence of the spinal cord upon the development of heat. In doing it we should have to enter upon the theory of fever, which is confessedly one of the most difficult parts of general pathology.

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[1] For further information see the following: *Wunderlich*, Archiv der Heilkunde. II. p. 547; and III. p. 175.—*Brodie*, Med. Chir. Trans. 1837. p. 416.—*Billroth*, Beobachtungsstudien über Wundfieber. 1862. p. 158.—*Erb*, Deutsches Archiv f. klin. Med. I. p. 175. 1865.—*Tscheschichin*, Reichert und Du Bois' Archiv. 1866. p. 170.—*Naunyn* and *Quincke*, Reichert und Du Bois' Archiv. 1869. pp. 174 and 521.—*Quincke*, Berl. klin. Wochenschr. 1869. No. 29.—*H. Fischer*, Centralbl. f. d. med. Wissensch. 1869. No. 17.—*R. Heidenhain*, Pflueger's Archiv. 1870. p. 578.—*Riegel*, Ibid. Bd. V. 1872. p. 629.—*Naunyn* and *Dubczanski*, Arch. f. exper. Path. u. Pharmak. I.

[2] Lancet, 1875, March 6, p. 340 (Clinical Society of London).

For the present it seems to us most natural to assume, with Naunyn and Quincke, that in these experimental lesions of the cervical cord, as well as in those of a pathological nature, there is a paralysis of certain paths which serve to restrict the production of heat. At the same time, however, an extensive vascular paralysis takes place, whereby an increased amount of heat is given off, which more or less compensates for the increased production. According to the preponderance of one or the other factor, the rise of temperature will be more or less considerable, or may be absent or even a minus quantity. Here much plainly depends on accidental external circumstances (temperature of the air, covering of the body, proportion of surface to weight of body, etc.). It is found, however, that an increase of the bodily temperature is most prominent in lesions of the cervical cord. The whole question deserves a fresh investigation, in view of the recent discoveries of Goltz respecting the vaso-motor centres in the cord.

The experiments cited have shown that in cases of section of the cord a *lowering of temperature* often occurs, especially where the effect of the vaso-motor paralysis is chiefly directed to the loss of heat from the surface. A similar condition exists in many diseases of the spinal cord; mechanical lesions (Fischer, l. c., Nieder [1]), chronic myelitis, the closing stage of tabes, etc. The temperature falls to 35°, 32°, 30° C. (95°, 90°, 86° Fahr.), or even lower; the patients meanwhile often live on for days and weeks. These are probably sometimes the temperatures characteristic of collapse, but at other times they are doubtless due to excessive loss of heat from vaso-motor paralysis.

6. *Disturbances in the Urinary and Sexual Apparatus.*

These are among the most important symptoms, for they always inflict great discomfort, and often influence the prognosis seriously. Our acquaintance with them, it must be admitted, is in many points defective.

a. Disturbance of the Secretion of the Kidneys.

But little is known of this at present, in spinal affections. In various spinal lesions, especially the more severe and acute forms, considerable changes in the character of the urine rapidly appear, but it is not yet clearly settled how far the kidneys and their innervation are directly concerned in this. After crushing of the cord by fracture of the vertebræ, after knife-wounds of the cord, after spinal apoplexy, in acute myelitis, etc., we often see the urine becoming turbid and slimy in a few days after the event, with blood and pus, with alkaline decomposition and its inevitable results, triple phosphates, and a horrible smell. It is generally thought that this change is caused in the first place by retention and ammoniacal decomposition of urine in the bladder, the result of which is a cystitis and an inflammation of the kidneys, secondary to the cystitis. Rosenstein[1] believes that he has obtained certain proof of this. But Charcot,[2] in view of the very rapid appearance of change in the urine, and the ecchymoses and foci of inflammation repeatedly found in the kidneys very soon after the spinal lesion, is forced to the conclusion that the spinal affection may be of itself the cause of the acute inflammation of the kidneys, and he lays special weight upon the element of irritation in these spinal lesions. Whether the case is similar in chronic disease, or whether in the latter the affection of the kidney is always secondary to that of the bladder, is likewise undecided at present.

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[1] Med. Times and Gazette. 1873. No. 1180.

Still less is known about anomalies of secretion, or extensive anatomical changes. The increased excretion of phosphates, observed in many chronic spinal cases, occurs in many other neuroses.

In respect to the alteration in the *quantity of urine* in spinal diseases very little information is furnished by human pathology. An analogy with the temporary suppression of this secretion, observed by Eckhard after section of the cord, may probably be found in a case of laceration of the cervical cord, observed by Brodie, in which the quantity of urine passed was extremely small. A considerable increase of the secretion sometimes occurs, a literal diabetes insipidus, accompanying spinal diseases (Friedreich, in degenerative atrophy of the posterior columns); it may be assumed with probability in these cases that the process has extended to the medulla oblongata.

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[1] Pathol. u. Ther. der Nierenkrankheiten. 2. Aufl. p. 287.

[2] Leçons sur les maladies du système nerveux. 1872–3. p. 117.

b. Disturbances of the Bladder and Changes in the Character of the Urine.

Spinal patients, without number, are attacked after a longer or shorter time by such disturbances, the occurrence of which always marks an unfavorable phase of the disease, owing to the difficulty of giving relief and the fact that they very often form the starting-point of the most serious complications.

These disturbances almost always begin with the paralysis of the bladder, so common in spinal complaints, and the consequent retention and stagnation of urine in the bladder.

In the chronic cases, which are the more frequent, and in which there is often no other complication than an incomplete and an infrequent evacuation of the bladder, decomposition of the urine occurs with slight alkalescence and deposition of concretions in the bladder; the consequence of this is a catarrhal cystitis, with formation of mucus and pus, abundant development of vibriones (which increases the facility of decomposition), an alkaline reaction, and a foul ammoniacal smell of the urine. Examination of the turbid urine shows a muco-purulent deposit, a few blood-corpuscles, numerous crystals of triple phosphate, vibriones, etc. The mucous membrane, at first in a state of catarrhal inflammation, is covered by degrees with erosions, is thickened and swelled, contains hemorrhages and pigment-deposits in its substance; the muscular coat of the bladder is hypertrophied, the wall of the bladder is thickened and retracted, etc. Purulent pyelitis and purulent disseminated nephritis very soon appear. This is the usual condition at the close of chronic spinal diseases (myelitis chronica, tabes dorsalis, etc.).

In quite acute cases the affection of the bladder not rarely opens with hæmaturia, with which are associated acute purulent or even ichorous cystitis, pyelo-nephritis, etc., very quickly leading to the most extensive decomposition of urine with all its consequences, high fever, uræmia, etc.

It remains to be proved whether the stagnation of the urine, produced by the vesical paralysis, is the sole cause of all these disturbances, or whether, as is extremely probable in acute cases, and is certainly possible in chronic cases, the lesion of the cord of itself constitutes a distinct cause of this inflammatory condition of the bladder and all its consequences. We know nothing certainly in respect to the nervous paths and centres in the cord which are concerned in these processes.

But it is certain that these conditions of the bladder may lead to the severest disturbance of the whole system, by the fever, the loss of fluids, and the retroaction upon the kidneys, which are associated with them.

c. *Disturbances of the Function of Urination.*

This very common and important class of symptoms comprehends many varieties, differing in their origin and course, as might be expected in the case of such complicated mechanism.

In the *chronic* cases, which are by far the most common, the first symptom is frequently a certain *difficulty in passing water;* the patient has to wait a longer time than usual, has to press more strongly in order to set the process going, and when the stream comes, it is small and slow, and at the close there is a more or less prolonged dribbling. Later, this increases more and more, and actual *retention of urine* occurs, which compels the regular use of the catheter, or may produce the "ischuria paradoxa," in which a distended bladder keeps up a constant dribbling. It is, however, possible that the retention may go on to the production of actual incontinence.

But, on the other hand, *incontinence* of the bladder may be the first symptom; the patient has to make haste the moment he feels the inclination to pass water; soon the discharge occurs at the same time with the inclination, and at the last it comes unexpectedly and involuntarily, is often quite unnoticed, and passes into the bed, the clothes, etc., at any time. The discharges may be considerable in quantity, and take place at certain intervals, or they may be frequent and small, or the urine may dribble constantly. Any of these disturbances may be further complicated by the appearance of cystitis.

In *acute* cases (sudden destruction or squeezing of the cord, myelitis acutissima, spinal apoplexy, etc.) complete retention usually exists from the outset, or within a few days. In many cases, as in experimental section of the cord (Goltz), it is merely the result of concussion of the entire cord, and depends on paralysis of the centres in the lumbar cord. Soon, however, the spontaneous (though not the voluntary) discharge returns, and passes directly into incontinence. The form which is assumed by the incontinence then depends essentially upon the seat of the lesion and the secondary changes in the cord. There either occurs, from time to time, a full, regular discharge of the bladder against the patient's will, and often without his knowledge—a sign that the reflex centre in the cord is in existence and the detrusor is not palsied—or there is ischuria paradoxa, an excessive fulness of the bladder with continual dribbling, in which case either the reflex centre is paralyzed and destroyed, or else the peripheral paths are interrupted. The detrusor is paralyzed together with the sphincter. The bladder is at first excessively distended, often reaching nearly to the navel; but subsequently, owing to catarrh of the bladder and hypertrophy of its wall, its dimensions diminish continually, though the incontinence continues unchanged.

Subsequently the symptoms may alter as partial improvement occurs in one or another of the nerve-paths; but the above description will be recognized as giving the ordinary traits of spinal paralysis of the bladder.

With our present knowledge of the mechanism of the evacuation of the bladder (see p. 54), it is not difficult to understand how the various forms of paralysis originate. It is, however, in special cases, often extremely difficult to decide which nervous apparatus is involved, as most of the symptoms may originate in more ways than one.

The complexity of these conditions will best be shown by a brief mention of the disturbances which may follow lesions of different portions of the paths which convey the innervation to the bladder. The evacuation of the bladder may be interfered with: 1, by lesion of sensory and motor peripheral paths; 2, by lesion of the reflex centres in the lumbar cord; and 3, by lesion of the sensory or motor paths which lead to the brain above the lumbar region.

If the peripheral sensory nerves of the bladder are alone paralyzed, the patient will probably perceive no further impulse to urinate, but will be able, from time to time, to pass water voluntarily by the action of the brain upon the centres in the lumbar cord; in this case the patient does not feel the evacuation. If the motor nerves of the bladder are peripherally palsied, the consequence will be retention with incontinence (ischuria paradoxa), in which voluntary urination is impossible. If both sensory and motor paths are paralyzed, as in lesions of the cauda equina, ischuria paradoxa, or at least absolute incontinence, will be the unavoidable result. (It is not yet established, whether the bladder, deprived of its spinal innervation, may continue to contract independently by virtue of the influence of the ganglionic apparatus situated in its walls.) All this holds good, of course, of the sensory and motor paths within the spinal cord previous to their connection with the reflex centres.

If these centres are paralyzed or destroyed, the necessary consequence will be complete retention followed by incontinence (ischuria paradoxa). The utmost that can be accomplished by the patient in such cases will be an incomplete evacuation by the aid of abdominal straining.

If the sensory paths on the other side of (*i. e.*, above) the lumbar cord are alone paralyzed, while the centres in the cord are uninjured, a regular discharge of the bladder will occur from time to time when it has reached the proper fulness; but the patient feels nothing of it, and therefore cannot prevent its occurrence. If only the motor paths above the lumbar cord are palsied, the patient can neither discharge his urine voluntarily, nor arrest, by voluntary contraction of the sphincter, an evacuation which has begun or is threatening; but he feels the inclination to urinate, which immediately excites a reflex evacuation of the bladder, withdrawn as it is from voluntary control. If all the paths above the lumbar cord are paralyzed, the periodical reflex discharges of the bladder take place without the patient's feeling anything of them, and without any power on his part to influence them. In most of the cases of this group the action of abdominal straining will not occur; but this does not much alter the symptoms.

We see that all the symptoms that have been described can be thus naturally explained, and it is necessary in each case to examine every circumstance with care, in order to ascertain the precise seat of the lesion. Such experience will soon convince us that the various forms of vesical palsy are very characteristically distinct; it will be easy in particular to decide whether the centres in the cord are in action or not, by irritating the wall of the bladder and producing reflex evacuation. Many cases, however, in which the disturbance is complicated and diffuse or ill-defined, will present insurmountable obstacles to exact diagnosis.

The localization of which we have spoken relates rather to ascertaining *at what height* in the spinal cord the nervous supply for the bladder is intercepted. We know, however, but little of the course taken by the paths concerned within the cord, and therefore are unable to say much about the location of a disease, relatively to the *transverse section*. The affections of the reflex centres must always be located in the gray substance of the lumbar cord; yet it should be remembered that a lesion of the root-fibres as they pass out may give rise to just the same disturbances as a lesion of the centres. In the case of the paths which lead to the brain, we must first think of the gray substance; in that of the motor paths, of the anterior columns also (Budge). Further information can only be given by special investigations.

The reflecting reader will find it easy to comprehend the course and the complications of those cases in which the alterations begin in a primary focus, and spreading from that gradually include other points in the conductive path; for example, when crushing of the dorsal medulla gives rise to myelitis of the gray substance, which extends downward to the lumbar cord and there paralyzes the centres for the bladder. It will also be easy to form a correct view of the processes of initial or slight disturbances of the functions of the bladder.[1]

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[1] In these statements we have intentionally avoided making a sharp distinction between palsy of the sphincter and of the detrusor, because, although conceivable theoretically, such a distinction hardly occurs in practice. We have reason to believe that the paths leading to both muscular systems, from the brain as well as from the centres in the lumbar cord, are close together, and therefore, as a rule, are affected

We have spoken only of spinal *paralysis* of the bladder; of its *spasmodic affections* in spinal disease little is known. As such, we ought perhaps to speak of numerous cases of excessive desire to pass water, or a few cases of ischuria. Nothing precise is known of these cases.

d. Disturbances of the Sexual Functions.

These have always been considered closely related to the spinal cord, and especially in the causal relation. They form, however, a very prominent portion of the symptoms of spinal troubles, and are much more noticeable in the case of the male sex, owing to the far closer dependence of the function upon the integrity of the cord in this sex than in females.

The symptoms in *men* suffering from spinal disease are:

Increased sexual desire and *increased sexual excitability;* every lustful thought, the sight, or the merest touch of a woman, causes erections. A state of weakness is usually associated with this; in coitus the ejaculation takes place too quickly; the erections mentioned are often immediately followed by ejaculation; pollutions by day and spermatorrhœa make their appearance. Whether there is really an *increased potency*, a power to practise coitus normally with unusual frequency, is questionable and is hard to decide, as, even under physiological circumstances, the sexual powers of performance differ extremely in individuals.

Far more common is the so-called *irritable weakness of the sexual organs*, which is especially observed at the beginning of spinal diseases and in functional debility of the cord. In this condition erections easily occur, which are weak, insufficient, and of short duration; the ejaculation during coitus takes place too soon, occurring before or immediately after intromission of the penis. The sexual sensations during coitus are diminished or quite wanting; the sexual appetite is lessened; the performance of the sexual act is followed by great exhaustion—a feeling of weakness, sweating, pain in the back, sleeplessness for several

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together. It will also be very easy to make out the cases of isolated palsy or weakness of the sphincter, which, properly speaking, is directly subject to the will alone.

hours, etc., and a feeling of great exhaustion usually lasting several days. Repetition of the act is impossible.

This condition is followed by *diminution* and complete *loss of potency*. The erections become continually rarer and weaker, are confined to the morning when the bladder is full, are usually absent when most wished for, and finally cease altogether. The sexual appetite usually disappears, but pollutions may occur with more or less frequency by day or night with or without sexual feeling ; they may, however, be entirely absent.

Priapism is not rare, in the form of frequent and continuous more or less complete erections. The power of copulation may be retained, and the sexual desire increased. Those cases are of more importance in which such pathological erections accompany a more or less complete interruption of conduction in the spinal cord ; they may originate in apparently a spontaneous manner, when the penis continues for a considerable time in a semi-erected state, more rarely in complete erection ; but more usually such erections are reflex, depending on external irritation, introduction of the catheter, friction of the skin of the glans or perineum or of the inner side of the thigh.

The investigations of Eckhard and Goltz furnish an easy and plausible theory of the manner in which these various disturbances originate under pathological circumstances, of the way in which interruption of the peripheral sensory and motor conduction, paralysis and irritation of the reflex centres in the lumbar cord, or cutting off or irritating the paths which pass from the lumbar cord to the brain, act upon the processes of erection, ejaculation, and copulation. It is not necessary to explain these in detail.

It may be added that the present state of our knowledge permits us to draw very few conclusions from disturbances of sexual functions, as regards the exact seat and nature of the spinal lesion.

As respects the disturbances of sexual function in *women* who suffer from disease of the cord, little is known. Ovulation, pregnancy, parturition may take place normally, even during severe spinal troubles.

In regard to the libido sexualis, and the act of copulation, nothing satisfactory is known.

7. *Disturbances of Digestion and Defecation.*

Of the *chemistry of digestion*, the *preparation* and *secretion of the digestive fluid* in diseases of the cord, almost nothing is known, though disturbances certainly occur. The secretion of the intestinal juices seems to be impaired in many cases, probably owing to the great tendency to constipation which often exists.

The *intestinal movements* are usually much affected, either in the way of increase or of diminution.

The former, of which the symptom is a frequent, watery-slimy *diarrhœa*, is the less common condition; it is often capable of being produced reflexly; thus, in a patient with chronic myelitis, I observed the regular evacuation of a mucous fluid mass from the intestine as often as his bed-sores were cleansed, and the like has been seen in dogs whose lumbar cord was cut.

Much more commonly there is habitual, often excessively obstinate *constipation*, of which almost all chronic spinal patients complain. The stool is slowly discharged, dry, and hard, and the evacuation occurs only at considerable intervals, and on application of energetic remedies. Several causes doubtless contribute to this—diminution of the intestinal secretion and of the peristaltic action, and probably also the weakness of the abdominal muscles of compression, which is often present. If there is an extreme degree of weakness, meteorism and accumulation of fæces occur, with their consequences.

We do not know exactly from what portions of the cord these disturbances proceed.

French authors, as Charcot,[1] Delamare, Dubois, and others, have described, in connection with tabes and other spinal affections, certain peculiar attacks, to which they gave the name of "crises gastriques," comprising violent pains, radiating from the back to the epigastrium, with uncontrollable vomiting, nausea, vertigo, etc. These attacks may last several hours or days; they recur periodically like the lancinating pains in the limbs of tabetic patients, and have manifestly a close analogy with such pains. They unquestionably depend upon transitory states of irritation in certain portions of the cord. I myself have repeatedly seen them in tabes

In like manner, there is occasionally observed (chiefly in tabetic patients) a violent and painful pressure in the rectum, conjoined with acute pains in the perineum, the anus, and the sexual parts. These symptoms also have, probably, a neuralgic character.

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[1] Leçons sur les maladies du syst. nerv. II. Sér. I. fasc. p. 32.

Of much more importance are the disturbances of *evacuation*, which accompany many diseases of the cord, and are analogous to a certain extent with the disturbances of the function of urination. These cases are chiefly caused by a paresis or paralysis of the sphincter ani, the consequence of which is a more or less severe incontinentia alvi.

In the mildest cases, the patients cannot retain their stool for any length of time, but are forced to yield to the pressure as soon as felt. This weakness may so increase that the discharge occurs at all times, and without obeying the person's will in the least. There may also be present a disturbance of sensibility, which makes the matter still worse; the patient does not feel the call, and even if he possesses some voluntary control, he is surprised by the discharge, of which he feels nothing, and is only informed of its occurrence by his nose or eye, or by the sensibility of his legs. This, we hardly need say, is a shocking infliction.

This paralysis of the rectum may, in acute cases, attain very rapidly to its fullest development, but in chronic cases it comes on very gradually.

It is easy to explain the several disturbances in the function of defecation, and their origin, by reference to the data of physiology (see above, p. 54). In brief, we would refer to what has been said under disturbances of urination, and in this place would only remark that our attention should be directed partly to the peripheral sensory fibres of the rectum and anus, partly to the motor fibres of the sphincter, and to the reflex centres in the lumbar cord, and the sensory and the motor paths which ascend from them to the brain. The action of abdominal pressure must also be thought of; and the ganglionic apparatus situated in the intestinal wall. The complexity of these points will increase the difficulty of an explanation, but in most cases we shall succeed in obtaining a satisfactory account of the nature, and probably also of the seat of the lesion. The remarks previously made in regard to disturbances of urination may be repeated here.

8. *Disturbances of Respiration and Circulation.*

Our knowledge of this branch of the subject is confined to isolated points, and is applicable only in a slight degree to the uses of pathology.

Disturbances of *respiration* occur in but few diseases of the cord, and almost exclusively in those of the cervical portion. The cord contains only conductor paths for the respiratory movements, situated probably for the most part in the lateral columns, and leaving the cord at various levels. The centres for respiration lie higher, in the medulla oblongata.

Lesions of the upper dorsal and of the cervical medulla, when they involve the lateral columns, always give rise to a disturbance of *inspiration*, which is severe in proportion to the height of the lesion. As long as it is confined below the point of exit of the phrenic, there is no danger, for only the intercostals and some auxiliary muscles of respiration are deprived of their function, while the diaphragm—the chief inspiratory muscle—keeps up the process of breathing to a sufficient extent. But, if the lesion seizes upon the roots of the phrenic nerve, the inspiration is always gravely impaired, even if only one side is attacked, while if both sides are affected, a fatal result from insufficiency of aëration is inevitable. Hence the rapidity with which death follows severe injuries of the uppermost part of the cervical cord, as in fracture of the odontoid process of the axis, etc.

In strictly unilateral lesions, disturbances of breathing will be seen, limited to the side of the injury in all cases.

It is much more usual to see disturbances of expiration in spinal disease, caused by paralysis of the expiratory muscles (of the abdomen and back). If the organs of respiration are sound, this gives rise to no great inconvenience; at the most, it becomes somewhat difficult to utter loud tones. But if bronchial catarrh and similar troubles exist, for which an energetic expectoration is required, the greatest danger to life may arise from accumulation of mucus in the bronchi. Hence the frequent fatal result of bronchitis, pneumonia, etc., in myelitic patients.

It is plain what must be the location of the disturbance in the spinal cord, in order to produce this difficulty in expiration.

The disturbances of circulation which accompany spinal disease have been very little examined, with the exception of the vaso-motor. They are confined to alterations in the action of the heart, which are seldom very great, as the influence of the spinal cord upon the heart is only subordinate. Nevertheless, changes in the activity of the heart seem not to be rare in spinal diseases, though little noticed. Charcot[1] notes a permanent *acceleration of the pulse* as a frequent symptom in ataxia; he also speaks of a permanent *retardation*[2] of the pulse as a noteworthy symptom of compression of the cervical cord, and describes it carefully.

The rapidity of the pulse can be influenced to a considerable extent, both by the sympathetic fibres which pass in the cervical medulla and by vaso-motor spasm or paralysis. If we further reflect that the root-fibres of the vagus and the spinal accessory descend to a considerable distance in the cervical cord, it will be clear that alterations of the rate of action of the heart are frequent in diseases of the cervical cord. The precise pathogenesis of these alterations must be ascertained in each individual case.

9. *Disturbances of the Oculo-pupillary Fibres, the Various Cerebral Nerves, and the Brain itself.*

We will here enumerate briefly a series of disturbances, of which only a part are directly referable to lesions of the cord, the remainder being more or less accidental complications, the connection of which with the spinal lesion is wholly unknown to us, even if it exists. But all these points may acquire such an importance in reference to the diagnosis of cases or varieties of disease, that it is quite worth while to mention them here, although we shall have to defer a minute examination to the special part of this work.

The connection between certain oculo-pupillary symptoms and spinal diseases is very clear. The fibres destined for the dilatator pupillæ originate in a centre situated in the medulla oblongata; they descend in the cervical medulla without decussation, and at various levels make their exit, passing to the cervical sympathetic, and thus to the eye. Irritation of these fibres produces *dilatation of the pupil* (mydriasis spastica), paralysis of the fibres causes *contraction of the pupil* (myosis paralytica). These phenomena may be unilateral or bilateral, according to the extent of the lesion in the cervical cord; in unilateral lesion the alteration occurs on the same side; the phenomenon is very characteristic, especially in lesions of one-half the cervical cord. Vaso-motor irritation or paralysis in the corresponding half of the face often accompanies the corresponding pupillary phenomena. A combination of the two constitutes a valuable symptom in diseases of the cervical cord. It should be added that, according to Robertson, Knapp and Leber,[1] the pupil in spinal myosis reacts to impulses of accommodation, but not to changes of light.

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[1] Leçons, etc. 2. Sér. 1. fasc. p. 56.

[2] Loc. cit. 2. fasc. p. 137.

The *hypoglossus nerve* is affected only in diseases of the cord which extend to the medulla oblongata; the resulting symptoms are paralysis of the tongue, disturbance of speech, and atrophy of the tongue.

The *vagus* and the *spinal accessory* seem not to be often affected; the consequences are spasmodic cough, dyspnœa, and anomalies of the action of the heart.

Still less is known of the affections of the *glosso-pharyngeus;* the paralysis of swallowing which occurs in many spinal diseases, especially in the later stages, is probably due to an extension of the morbid process to the paths of the glosso-pharyngeus, which lie in the medulla oblongata.

The *auditory nerve* is now and then attacked (*e. g.*, in tabes); the connection between the atrophy of the auditory nerve and the spinal disease is entirely obscure. Nervous deafness, or loss of hearing for high or low notes, is then observed.

The *facial nerve* is very rarely affected in spinal disease; the lower branches are more liable. The usual cause is extension of disease to the medulla oblongata.

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[1] Virchow-Hirsch's Jahresbericht für 1872. II. p. 544.

Symptoms from the *trigeminus* are much more common; the sensory fibres are oftener attacked, the motor more rarely. The symptoms are formication, anæsthesia, pain; for which the affection of the upper cervical cord is a sufficient explanation.

The involvement of the muscular nerves of the eye is very common in spinal diseases, and very hard to explain. It is especially common, in the stage of precursory symptoms in tabes, to observe paralysis of one of these nerves, either in one eye or in both. The oculo-motorius is most often attacked, next the abducens, less frequently the trochlearis. We have at present no right to speak of this disease as depending on the spinal lesion; we know nothing of a trophic action of the spinal cord upon the cerebral nerves; we are forced to assume that the degenerative atrophy is localized simultaneously in several points of the cerebro-spinal axis.

The same is true of those extremely common affections of the optic nerve, which, associated with tabes dorsalis, add horrors to a disease already sad enough. These cases are always due to progressive gray degeneration of the optic nerve, recognizable by the increasing atrophy of the papilla. Amblyopia, color-blindness, contraction of the field of vision, are the first symptoms, and lead with frightful rapidity to total amaurosis. Similar affections of the optic nerve, not quite so hopeless as to prognosis, occur also in multiple sclerosis. The connection between this disturbance and the spinal disease is wholly unexplained; the amaurosis often precedes by many years the first appearance of the tabetic symptoms (lancinating pains, anæsthesia, ataxia).

Of affections of the *olfactory nerve* in spinal diseases, nothing is known at present.

A good deal of research will be required in order to establish upon an accurate basis the connection between these diseases of cerebral nerves and spinal disease. If we refuse to localize the disorder in several spots at once, we shall be most likely to assume a propagation of the process to the nerve-nuclei in the medulla oblongata, and perhaps a spreading of meningeal processes at the base of the brain; probably other relations, as yet unknown, will hereafter be discovered.

Concerning the *disturbances of speech*, which are not rare in spinal affections, a few words will suffice. These are never psychical (aphasia proper), but probably are solely peripheral and motor in character, originating in the muscular apparatus of speech (*anarthria*). This may be due to paralysis of the hypoglossus, which will injure the lingual sounds, or of the facialis, which interferes with the labials, or of the velum palati, which gives a nasal tone to the voice, or, finally, of the accessorius, which interferes with the formation of the voice, and may produce aphonia. Not infrequently a sort of ataxia, an inco-ordination of the movements of speech, an irregular stuttering way of speaking, is observed, as in many cases of tabes; and finally, a slow, scanning speech is highly characteristic of multiple sclerosis. Many other disturbances of speech occur, of more or less importance.

The brain itself may be involved in the spinal disease in a great variety of ways and in very various degrees. It is characteristic of many cases of spinal disease, that the brain remains intact with its functions of intelligence, memory, capacity for work, etc., and that the cerebral nerves are not in the least affected. But in other cases, not less characteristic, we observe a more or less general implication of the brain in the morbid disturbance. This may occur in several ways, *e. g.*:

(*a*) The same process may be localized both in the brain and in the cord, or it may extend from the latter to the former; thus, in multiple sclerosis, there may be sclerotic patches in both brain and cord; in tabes, gray degeneration of the posterior columns of the cord and of the optic or other cerebral nerves; in dementia paralytica, simultaneous degeneration of the cord; the same is true, in syphilis of the central nervous system, in meningitis cerebro-spinalis, in meningitis tuberculosa, paralysis ascendens acuta, secondary descending degeneration of the lateral columns in consequence of cerebral affections, etc. In every case the cerebral symptoms here form important features in the *ensemble*.

(*b*) Cerebral symptoms may further be caused by the secondary effects of the spinal disease; as by uræmia due to cystitis and nephritis, by pyæmia due to decubitus, etc.

(*c*) Finally, severe cerebral symptoms occur in a way not yet understood in the final stages of many spinal disorders; as delirium, coma, excessive temperature, spasmodic conditions, with which cases of tabes or chronic myelitis often terminate. It is hard to say how these symptoms originate; usually the excessive cachexia, due to the spinal disease, may be the proximate cause; but may it not be supposed that vaso-motor actions originating in the cervical medulla may change the circulation and nutrition of the brain, and thus form an intermediate member between the spinal disease and the cerebral symptoms?

We have now enumerated nearly all the forms of disturbance which occur in connection with the spinal cord, and have endeavored to develop their pathogenesis, as far as possible, in order to give the practical physician a scientific view of the connection between apparent diseases and the fundamental lesions in the cord.

The coalition and varied grouping of single symptoms constitute the characteristic forms of disease which we meet in practice. Daily experience serves to show that the greatest variety exists here; sometimes we meet with a combination of motor and vaso-motor disturbances, and sometimes one of sensory and motor disorders; to these are superadded in various instances alterations of reflex function, or anomalies of the function of the bladder and sexual organs; in other cases single cerebral nerves are implicated in the disorder, or the trophic disturbances assume a leading place, etc. This multiplicity of symptoms permits us to divide them into certain groups, many of which are already well-known forms of disease, while others stand in need of a stricter definition.

The exact localization of the several disturbances is often significant; for example, when both the lower or only both upper extremities are attacked by palsy, when anæsthesia or pain exists at a certain level in the trunk or limbs, when the motor palsy is confined to one side, and the sensory to the other side of the body, etc.

It is the object of clinical study to ascertain what is characteristic and common in all these symptoms; having done which, we are to draw sharply-defined portraits of disease, and to refer the latter to changes in the cord, strictly defined as to locality. In the special part of this work we shall see how far the pathology of the day has gone in solving this difficult problem.

B. General Etiology of Diseases of the Spinal Cord.

The etiology of diseases of the cord is not in a very satisfactory condition. A great many isolated facts have been recorded, not always sufficiently well founded, but the really scientific results are small, few general points have been established, and the pathogenesis of the forms of disease remains obscure in most cases.

We must therefore content ourselves with brief outlines, leaving the fuller details to the special part. We will state here only that which is in some degree certain, but the most part of our remarks will relate to defects in our knowledge.

First, there is a series of causal agencies and injuries which place the cord in a condition of increased susceptibility to disease; these may be designated as *predisposing causes*. They are certainly very prominent in the pathogenesis of spinal diseases, but we should not forget that the same injuries may under certain circumstances produce not only the morbid tendency, but also the disease itself; and that, therefore, they may in some cases become direct causes. This depends partly on the intensity with which they act, partly on the accidental combination of favoring circumstances.

One of the most powerful predisposing agencies is doubtless the so-called *neuropathic disposition*, that peculiar disturbance in the nutrition of the nervous apparatus which lessens the resistance of the latter to all possible injuries, and increases the tendency to disease in a definite direction. It may exist in the spinal cord, and predispose it to pathological reaction under all kinds of irritation. Numerous spinal diseases are, without doubt, referable to this condition.

This neuropathic condition is certainly congenital in most cases, and is transferred from parents to children. Whole families, many generations, may be thus burdened with neuropathy, and cursed with a feeble power to resist, on the part of the nervous system. In many cases the effect is seen in a general neuropathic disposition; all possible neuroses (hysteria, tabes, epilepsy, psychoses, etc.) are at home with the members of a family, and each one is affected in only that portion of his nervous apparatus which is accidentally the subject of a special injury. It is not at all necessary that the parents should have suffered from the same disease; the children, in such cases, inherit merely a general disposition of the nervous system to disease, which may be manifested in very various ways, according to the nature of the incidental causes.

In other cases a perfectly definite disposition is inherited by the cord, so that children are attacked by the same affection as their parents (direct inheritance), of which progressive muscular atrophy furnishes the best examples; or it may happen that several or all of the children of one couple suffer from the same disease when neither parent has had it, as in the cases of degenerative atrophy of the posterior columns, published by Friedreich.[1]

The way in which this neuropathic disposition is propagated from generation to generation, often with increasing intensity, is entirely obscure; and we are quite ignorant of what the finer changes in the nervous system, and especially in the cord, may be.

The exaggerated susceptibility to disease on the part of the nervous system, and especially of the cord, may also be *acquired* by a variety of circumstances which must be reckoned among the predisposing causes.

Of these, *sexual excesses* and *irregularities* occupy the first place. Their effects were formerly often overestimated; but at the present time the tendency of opinion is so strongly in the other direction that an author like Leyden makes no mention of them in his general etiology.[2]

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[1] Virch. Arch. Vols. 26 and 27.

[2] Klinik der Rückenmarkskrankheiten. I. p. 170.

My own opinion, based upon observations which for some time past have been specially directed to this point, is that these causes are decidedly important in the production of numerous spinal diseases—a view which is represented in the writings of many prominent authors (Romberg, Nasse, Hammond, Salomon, M. Rosenthal, and others).

I believe we may say that *any gratification of the sexual passion, whether natural or unnatural, indulged in to excess and for a long time, forms for many men—not for all—a circumstance that powerfully depresses the spinal cord, and predisposes it to disease.*

The processes which accompany the sexual act, especially ejaculation, are attended with a very violent exaltation and shock of the entire nervous system; and the spinal cord seems to be the organ which suffers most.[1] Certainly this element seems to us of far more importance than the comparatively trifling amount of loss of material which occurs in the discharge of semen.

The facts may be analyzed as follows:

Excessive natural coitus, in many persons, certainly produces symptoms which point to a weakness and a diminished functional capacity on the part of the spinal cord; weakness of the legs, inability to stand for a long time, trembling when forcible movements are made, pains in the back, shooting pains in the legs, sleeplessness, etc. This may often be noticed in the newly-married, or in persons who have indulged in great excess for a short time. If the cause of these symptoms soon disappears, the injury may in most cases be quickly repaired; but if the excesses are continued, further injury, or even positive disease, occurs. Any external injury, exposure to cold, excessive walking, etc., may then bring on the worst results.

It is really hard to say at what point *excess* begins. No number can be given; the disparity in the powers of different men is enormous. While for some men Luther's rule, "die Woche zwier" (twice a week) denotes the limit of their capacity, others can with impunity do four, six, or ten times as much. This seems to depend on congenital differences in the sexual power, such as is found also in animals (stallions, etc.). A slight degree of potency seems to me to be a common circumstance among numerous members of nervous families. Of course such a weakness may be acquired by all sorts of influences which depress the nervous system. In determining the question of excess we must, therefore, take pains to ascertain the powers of the individual.

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[1] *Wundt*, Physiologie. 2d edition. p. 690.

In very young people, not fully grown, and in elderly persons, the evil effects of excess are more readily perceived than in persons of the vigorous age. If coitus is begun at a very early age, and practised with excessive frequency, its evil consequences follow with more or less rapidity in the form of spinal weakness, general nervousness, etc. Youth has immense power to repair losses, but the consequences of early squandering of power often appear later in life.

A specially injurious effect upon the spinal cord is ascribed by many physicians to coitus practised in the erect posture; it is often given as an occasional cause of acute spinal disease.

The effects of *unnatural gratification of the sexual appetite —onanism*—are exactly the same as those of the natural indulgence. The consequences are much exaggerated, but nevertheless they do exist, and are much more pronounced in the case of certain individuals—weakly, irritable, nervous persons—than in that of others. Onanism, commenced early, practised frequently, and continued for years, must be most decidedly injurious. The undue irritation of the nervous system which it causes, occurring in the period of growth and development, is seldom without injurious results, of which the most prominent consist in weakness and irritability of the nervous system.

It is common to consider onanism much more dangerous than natural coitus. This does not seem credible to us. The effect upon the nervous system of a man must be the same, whether the friction of the glans is effected in the vagina of a female or in some other way; the nervous shock of ejaculation is the same; it might even be naturally supposed that the nervous excitation would be greater in case of connection with a woman. But the frequent repetitions of excitement at an early period of life, which are caused by self-abuse, are certainly very dangerous; and it is furthermore quite certain that the feeling of degradation—so commonly felt, and so well grounded—the perpetual struggle between the powerful impulse and the moral duty, must wear and exhaust the nervous system. These circumstances may increase the evil effects of onanism. But it is only the *excess*—that which for the individual is excessive—which does harm; if practised to a moderate extent, onanism is no more dangerous than natural coitus. There are not a few men who are unable to obtain the natural indulgence owing to circumstances, or who are

afraid of contagion, or who think onanism less degrading than contact with public prostitutes—who, from time to time, practise onanism, certainly without injury to their health. Of the moral effects of this vice this is not the place to speak.

A similar effect is produced by *habitual pollutions*, if frequently repeated for years together. They are especially common in onanists, and are rather to be taken as evidence of an existing disorder, than as liable to become the cause of such. But even in such cases they often aggravate the difficulty.

Finally, in delicate and nervous persons, very destructive effects upon the nervous system are produced by long-continued *sexual excitement without gratification*, such as often occurs in prolonged and very affectionate courtship.

All this is applicable only to the male sex. In the female, very little is known of this matter, and it is of course very difficult to obtain information. I have never found that public prostitutes exhibited any special tendency to spinal diseases.

The effects of sexual excesses find a parallel in all sorts of influences which give rise to an *undue strain upon the nervous system*, and *especially the spinal cord*. They produce exhaustion and over-stimulation more or less quickly, and thereby increase the danger of disease. Among such circumstances may be named excessive bodily efforts, marching, climbing of mountains, riding, etc., especially when the supply of nourishment is deficient, and sleep is insufficient; also protracted watching by night, deprivation of sleep, violent and continuous excitement of passions, and, without doubt, also excessive mental efforts, especially when combined with other injurious agencies, such as severe bodily exertion or sexual excesses.

To the *period of life* only a slight predisposing influence upon certain spinal diseases can be assigned; such diseases occur at all ages. In a few, however, there exists a decided preference for childhood, and in others for adult or advanced life. The tendency to spinal diseases is certainly greater in adults, as will be shown in the special part of this work.

Sex has still less predisposing influence than age. There certainly are diseases which occur much oftener in men than in women, as tabes, but this may be explained by the fact that men are much more exposed than women to certain forms of injury.

To *general disturbances of nutrition*, on the other hand, of the greatest variety of character, we may ascribe a decided predisposing influence; all states of anæmia and cachexia depress the nutrition of the cord, simultaneously with that of the general system, and increase its susceptibility to morbific agencies. Thus may be explained the effect of loss of blood, chronic disturbances of digestion, severe and protracted acute diseases, long-continued loss of fluids, etc.

Among the *incidental causes* of spinal disease, the simplest and most direct are without doubt those of *traumatic* origin. There are numberless ways in which this may take place directly; cases are known of gunshot, stabbing, incised, and other wounds, crushing and destruction of substance by fractures or dislocations of the vertebræ, shocks from severe falls or railway collisions (railway spine of the English), and so forth. No explanation is needed of their mode of action, or of their consequences (inflammation, softening, necrosis, degeneration, etc.).

In close relation to these causes stands that of *slow compression* by pathological growths, by tumors, abscesses, new formations, exudations, curvature of the vertebræ, etc. Inflammation, secondary degeneration, etc., are of frequent occurrence in these cases.

Direct propagation of neighboring morbid processes forms an equally evident source of spinal disease. Thus, inflammation and suppuration of the vertebral bones or the adjacent soft parts may extend to the membranes of the cord, or to the cord itself; new growths may intrude upon it, the gangrenous inflammation of bed-sores may seize upon the contents of the spinal cord, etc.

But the effect of *exposure to cold*, evident as it is in fact, is quite obscure in its nature. Nothing is more certain than that, in a very great number of cases, a sudden or continued cooling of the surface of the body is followed by spinal disease. This has been seen after a fall into the water, after sleeping on the damp ground, sudden drenching of the clothes or exposure to a draft when the body is heated, bivouacking in snow or rain, working in ice, in damp cellars, in cold water, etc. There are several diseases which may be produced in this way, spinal meningitis, myelitis, tabes, spinal palsy of children, tetanus, etc.

Of the activity of this cause there cannot be the slightest doubt: its effects seem most decided in nervous, irritable persons with predispositions to spinal disease, or in cases where other injurious agencies, as great bodily exertion, mental excitement, etc. (as in military campaigns), have come into play.

But the manner in which these circumstances act is still entirely unknown; it is probable that the morbific influence consists in a reflex action, originating in the skin. But we have only hypotheses as regards the way in which this influence produces inflammations and other disturbances of nutrition in the cord. It is not yet certain whether a cooling of the blood has a share in the result—the lower temperature of the blood being supposed to act as a direct stimulus to the cord. It is hard to conceive how the direct action of cold should affect in any other way an organ so deeply placed as the cord, although myelitis can be produced by the application of severe cold to the exposed cord.

We are still wholly in doubt as to the reason why the same cause produces in one person tabes, in another myelitis of the gray anterior cornua, and in a third, meningitis or tetanus.

Disturbances of circulation, of very various origin, may cause sundry disturbances of the spinal cord; hence the effect of suppressed menses, of hemorrhoidal disease, of arterial fluxions and venous congestions, of vaso-motor disturbances, of embolism and thrombosis, atheroma of the spinal arteries, etc.

A frequent cause of spinal disease consists in *excessive exertion* of any sort, exhausting the cord. In this category are included sexual excesses, when practised frequently and at short intervals, walking, riding, swimming, or other muscular acts which go beyond the natural powers. All these may become the point of origin for severe spinal disorders, especially when they affect predisposed individuals, or when other agencies coincide—as that of cold; whence the frequency of these affections after laborious campaigns, bivouacs in winter, etc.

Psychical influences are a less frequent agency in the production of spinal disease. Fright, alarm, disgust, etc., seem to have a pretty clear connection with the origin of general and diffused neuroses (epilepsy, chorea, hysteria, and the like); but not to the same extent with that of spinal diseases. But there are some cases in which paralyses and other disturbances of a probably spinal nature have been seen to arise from purely psychical states, especially fright and terror. Thus, J. Russell Reynolds[1] observed the occurrence of paraplegia in a young lady, arising from a fear of the disease; she was nursing her father, who was paraplegic. Hine[2] saw an acute and fatal myelitis in a pregnant woman, caused by violent emotion. Leyden reports a case of paraplegia caused by fear at the breaking out of a conflagration, and Kohts[3] tells of similar occurrences at the bombardment of Strasburg. It is quite uncertain how we ought to interpret these facts, and whether the psychical emotions act through the vaso-motor paths,[4] or whether they may directly produce a disturbance of the finer processes of nutrition in the central nervous elements.

Certain *intoxications* present well-known spinal symptoms; such are poisoning with strychnia, arsenic, phosphorus, lead, etc. Some of these poisons seem to have the power, when long continued, to produce marked spinal diseases; for example, lead.

The local development of various infectious diseases, acute or chronic, is of great importance in etiology. Syphilis, by becoming localized in the vertebral column, the membranes, or the cord itself, may lead to spinal symptoms; tuberculosis not seldom attacks the cord and its membranes; and of the acute infectious diseases there is one (meningitis cerebro-spinalis) of which the chief seat is the pia mater cerebro-spinalis. To these must be added the cases, not few in number, of spinal affections *following acute diseases* (typhoid, acute exanthemata, intermittent, influenza, pneumonia, etc.), which, as a rule, must be regarded not as a specialized localization of the original process, but rather as somewhat accidental complications of the latter, dependent on a local predisposition established by the acute disease.

A very frequent source of spinal disease is found, lastly, in

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[1] Remarks on Paralysis, etc., Dependent on Idea. Brit. Med. Jour. No. 6. 1869. p. 483.

[2] Med. Times. 1865. Aug. 5.

[3] Berl. klin. Wochenschrift. 1873. Nos. 24-26.

[4] In the third case of *Kohts* the menses were instantly suppressed by fright.

irritation and disease of peripheral organs. Of this we have already numerous proofs. Paraplegia has been seen with especial frequency following severe and obstinate dysenteries and other intestinal diseases, and chronic affections of the kidneys and bladder ; in many cases the autopsy has shown myelitis to be the cause of the paraplegia. Such occurrences have been less frequently observed in uterine disease, which is more commonly followed by hysterical palsies—though the latter also, in some cases, are certainly of spinal origin. Myelitis has also been observed to follow peripheral lesions of nerves, diseases of joints, and so forth ; the tetanus which follows injuries of nerves and peripheral lesions certainly belongs to this class.

All these processes have long been studied with great zeal; they have, in fact, been made into a special class of *reflex disorders* (usually termed *reflex* paralyses), because they have been commonly supposed to originate in the reflex way from peripheral irritation. But the theory of these reflex diseases is in dispute to this day; and the number of works written upon them is very large.[1] We have discussed this point in another place;[2] the statements there made relate chiefly to reflex paraplegia and the myelitis which causes it, and we may therefore refer to that place, to avoid repetitions. The spinal disturbances which follow peripheral irritation or disease cannot, therefore, as a general thing, be referred to a purely reflex disturbance of function; they must, for the most part, be dependent on coarser changes of nutrition (inflammation, softening, exudation) in the cord. Regarding the connection of the latter with the primary irritation, we are not yet fully in possession of the facts ; it is in part effected by an ascending neuritis, of the existence of which there is no doubt; but in other cases the inflammation is transmitted to the cord in the reflex way.

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[1] For more minute information we refer the reader to the following : *Leyden*, Ueber Reflexlähmung. Volkmann's Sammlung klin. Vortr. No. 2. 1870.—*Lewisson*, Hemmung der Thätigkeit der motorischen Nervencentren, etc. Reichert u. Du Bois' Archiv. 1869.—*Feinberg*, Ueber Reflexlähmung. Berl. klin. Wochenschrift. 1871.—*Tiesler*, Ueber Neuritis. Diss. Königsberg. 1869.—*Brown-Sèquard*, Lectures on the Diagnosis and Treatment of the Principal Forms of Paralysis of the Lower Extremities. London. 1861.—*Jaccoud*, Les paraplégies et l'ataxie du mouvement. 1864.—*W. Gull*, Med.-chir. Transact. Vol. 39. 1856. p. 195.

[2] See Vol. XI. p. 399.

A few attempts have lately been made to settle this question, but without much success. The investigation by Roessingh[1] threatens to set us back a step, as he has arrived at entirely negative conclusions in his repetition of the experiments of Lewisson and Feinberg.

Klemm's[2] laborious studies leave room for many questions and doubts. They by no means prove that a direct propagation of the inflammation takes place along the nerve to the central organ: an extension *by leaps* was all that was proved. In this case, therefore, and especially where inflammation is transmitted to the symmetrically situated nerve of the opposite side of the body, without demonstrable affection of the central organs, the only explanation is that which assumes a sort of reflex transmission. The process may be essentially like that in which exposure of the skin to cold produces inflammation of the spinal cord. In so-called "reflex paralysis," the irritation is of another sort, and acts upon some other organ than the skin.

The question of reflex disease of the cord is certainly still in doubt, and is in great need of further clinical and experimental study.

C. General Diagnosis of Diseases of the Spinal Cord.

When we encounter a complicated nervous affection, the first step to be taken is a careful enumeration of the existing disturbances. The first and most important point is, to test all the departments of the nervous system, and thus to ascertain the existence, the grouping, the succession, and the history of each symptom. The diagnosis is made from the total of all these.

The next question is always that of the *location of the disease;* the organ affected; in nervous diseases, the choice lies between the brain, the medulla oblongata, the spinal cord, the peripheral nerves, and the sympathetic.

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[1] Bijdrage tot de Theorie der Reflexparalyse. Nederl. Tijdschr. vor Geneesk. 1873. Bd. I. No. 53. See Virchow-Hirsch's Jahresb. for 1873. Bd. II. p. 44.

[2] Ueber Neuritis migrans. Diss. Strassb. 1874.

The reply to this question, in the case of the spinal cord, often involves great difficulties. We may, it is true, follow the good old rule, to place the point of lesion at a spot where all the affected paths lie nearest together. But this rule often goes but little way with the spinal cord, both because all the paths which it contains pass into the peripheral nerves, and may be diseased in them, and also because a plurality of seats of disease is possible, and, in the case of the central nervous system, is very common. There is no function, quite specifically peculiar to the cord, the impairment of which would lead us at once to recognize an affection of the cord: this statement is true, even in regard to the disturbances of reflex activity.

When, therefore, disturbances of the sensory and motor system, of the vaso-motor and the reflex functions, of the trophic conditions and of the genito-urinary functions, etc., coexist, and in parts which directly depend on the cord for their innervation, then there is a *great probability* that the cord is affected; but *certainty* does not exist until we have excluded the affections of the peripheral paths. This is certainly possible in many cases, but not in all; there are, for instance, diseases of the cauda equina, which cannot be distinguished with certainty from those of the cord, and the same is true of extensive disease of the nerve-roots, etc.

In such dubious cases, various means may be employed to confirm the diagnosis, such as the data of the history of the case, or the causal factor, which often give us an opportunity to infer a definite seat of lesion.

By far the best aid is that furnished by *experience*, which teaches us that certain well-characterized groups of symptoms correspond to very definite lesions of the cord. We are in possession of a series of groups of this sort, which can without hesitation be recognized as dependent on disease of the spinal cord; thus, tabes dorsalis, the so-called acute spinal paralysis of children and adults, sclerosis of the lateral columns, progressive muscular atrophy, tetanus, and many others.

Experience goes still further; it often gives us notice by a few symptoms, often by a single one, of a threatening or actually present disease of the cord, because the constant or almost constant, coincidence between the symptom and the disease has become established through observation; thus, for instance, tabes may often be recognized by a precursory atrophy of the optic nerve, or by lancinating pains.

It therefore follows that in order to form a correct and sure diagnosis of a spinal disease, we need not only a very careful and comprehensive investigation, not only an accurate statement and estimation of the etiological and other elements, but also an intimate acquaintance with the whole of the pathology of the cord, and a good bit of practical experience.

There remain, however, a few cases in which the diagnosis may be difficult, and in which the spinal location of the disease is not quite certain. It then becomes necessary to distinguish the spinal from the peripheral disease, on the one hand, and from the cerebral on the other—which is often very difficult. We must, however, here confine ourselves to naming a few points which will assist diagnosis in given cases.

In favor of a peripheral localization, the following circumstances may be mentioned: Limitation of the disturbances to single nerves or branches of nerves; exact coincidence of the motor, sensory, vaso-motor, and trophic disturbances with the distribution of a peripheral nerve; absence of retarded conduction of sensation; absence of all reflex action; absence of weakness of the bladder and the sexual functions, etc., in case the sacral nerve itself is not the seat of disease; presence of great disturbance of the trophic function; certain results of electrical examination;[1] the existence of a known local cause of lesion.

In favor of a cerebral location we may have the following (among others): hemiplegic distribution of the disturbances, with the sensory and motor disturbances upon the same side; unequal intensity of the sensory and the motor disturbances; absence of all trophic disturbance; entirely normal electric reaction; retention or exaggeration of all spinal reflex acts; retention of associate movements and automatic movements, and of the rectal and vesical functions; presence of disturbance of the higher senses, and of various cerebral nerves (except so far as well known to be often involved in spinal disease), of disturbances of speech and of the mental functions; finally, the presence of headache, giddiness, and causeless vomiting.

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[1] See Vol. XI. p. 445.

In favor of the *spinal seat of disease*, the following circumstances may be used in evidence: the usually paraplegic distribution of symptoms; crossing of motor and sensory disturbances in case of hemiplegic symptoms; sensation of a girdle at the upper limit of the other disturbances; change in a portion of the spinal reflex acts (exaggeration or weakening); weakness of the genito-urinary functions; paralysis of the rectum; trophic disturbances, bed-sores, etc.; definite paræsthesiæ, retardation of the conduction of sensory impressions; disturbance of certain automatic movements; the peculiar local limitation of spasms of cerebral origin; absence of psychical changes, and usually, of disturbances of the higher organs of sense and the cerebral nerves.

It must also be noted that all these points possess by no means an absolute, but only a very conditional value; that their significance is very often not decisive, except in connection with many other symptoms, so that they cannot be made useful for diagnostic purposes except after a very careful estimation of all circumstances.

When we have decided that the cord is the seat of disease, we have to proceed to *localize the lesion within the cord.* In doing this, the distribution of the symptoms, especially those of paralysis, usually furnishes an excellent point: it is often possible to decide within a hair's breadth to what height in the cord a certain affection extends, and the gradually upward progress may often be followed in a very beautiful manner. While thus the upper limit of a lesion is usually very easily recognized, the same is not equally true of the lower limit, and it is often hard to decide whether the lesion is diffuse or circumscribed as respects the longitudinal section of the cord. Yet there are certain points which indicate that the lower portions of the cord are intact; the principal of these (see General Symptoms) are those relating to the reflex function, that of the bladder and rectum, and the nutrition of skin and muscles.

This is the case when disease extends over the entire transverse diameter of the cord, and to some extent, also, in the longitudinal direction of the cord.

We are enabled by experience to recognize also diseases confined to limited parts of the transverse section; such may also extend to a greater or less distance in the longitudinal axis. Thus we can distinguish the affections of the separate white columns, the anterior and the central gray matter, etc.; those of the white posterior columns (probably only the outer divisions) furnish the symptoms of tabes dorsalis (see the special part); that of the white lateral columns, the symptoms of Charcot's lateral sclerosis (which see); that of the anterior gray cornua in its acute form, the symptoms of infantile spinal paralysis; in its chronic form, probably those of progressive muscular atrophy; disease of one lateral half of the cord, symptoms of Brown-Séquard's unilateral lesion; disease of the central gray substance gives rise to an equally characteristic group, and in general, the implication of the gray substance may be recognized by the disturbances of nutrition, of reflex action, of electrical reaction, etc.

Thus, in many cases, it is possible to decide very accurately with regard to the seat and location of the lesions in relation to the longitudinal and transverse sections of the cord; it is certain that the perfection of the methods of anatomical research, recently applied to the diseased cord, will soon add much to our means of diagnosis. And a good deal remains to be done, for there are considerable parts of the transverse section, the lesions of which have never yet been brought into relation with any group of symptoms.

It remains for us to decide the *nature of the lesion*—whether paralysis or irritation, inflammation or degeneration, softening or atrophy and sclerosis, compression or bleeding or other lesions, are present in the cord.

It is difficult to establish general diagnostic rules for this; symptoms of irritation (spasm, pain, increased reflex action) will incline us to infer a corresponding pathological state; symptoms of paralysis will point rather to degenerative processes, softening or compression and destruction of the cord; but we must be cautious in these inferences, since both sorts of symptoms and both pathological conditions are very often combined, and the same disease in its progress not seldom leads to a manifold alternation of symptoms.

But, as a rule, more and better points can be derived from the results of experience, from the development and order of succession of the symptoms, from the history of the case, the etiology, the objective examination, etc.

It would lead us too far to attempt to give and analyze examples of even a portion of the possible cases here alluded to. We shall find opportunity for doing this in the special part of this work. Our present object is confined to indicating the points for diagnosis, and the methods and instruments, the precautions and the care, required in order to render the diagnosis exact. As regards complications, we will only say, in brief, that they must be ascertained and judged according to the usual diagnostic rules.

IV. General Therapeutics of Diseases of the Spinal Cord.

This branch of our subject presents many weak sides. In most diseases of the cord, our success in the use of remedies is rather small. The general opinion regarding spinal diseases, that they are nearly or quite incurable, is but too well founded.

It is true that we have recently had to modify this view essentially. A great number of curable diseases have been referred to a spinal origin; and, on the other hand, the prognosis in many chronic spinal diseases has been much improved by the progress of therapeutics.

Yet much remains to be done. And above all, we must learn how to recognize the diseases, before we can treat them rationally; we are still at the threshold of exact knowledge of the pathology of the cord, and the scientific therapeutics of its diseases is still in its first stage.

The attempt to state the general principles of treatment at this day seems hazardous, so small is the material, and so uncritically reported. The attempt will nevertheless be made to present the remedies which are used in disease of the cord, and especially those which we have reason to suppose exercise a decided influence over that organ. For the present, we must be content with such a mere attempt.

The indications in spinal disease are very numerous, consisting of, *a*, the removal of so-called functional disturbances (intangible disturbances of nutrition), especially those of a chronic sort ; *b*, alteration of disturbances of circulation (hyperæmia and anæmia) ; *c*, the cure of acute anatomical changes (acute inflammation, softening, hemorrhage, etc.) ; and, finally, *d*, the removal of chronic anatomical changes (degeneration, atrophy, sclerosis, induration, new formations, etc.).

All the usual methods of treatment are, of course, employed for these objects, with suitable modifications to adapt them to the seat of the disease ; the functional disturbances are remedied by regulation of the function, by slight stimulation of the latter, by alteration and improvement of the nutrition and sanguification ; for disturbances of circulation, we have a variety of means for acting on the vaso-motor system and the vessels ; acute inflammatory processes are treated by antiphlogistics, derivation, etc.; the chronic alterations are usually attacked by alterative, exciting, and derivative procedures. In the cure of these chronic cases nature has, of course, the chief part to play ; we have only to supply the most favorable conditions for repairing the disorders, to excite the desired change in the general nutrition by certain remedies, by promoting the formation of blood and the processes of nutrition, by stimulating the metamorphosis of tissue, by regulating the function of the diseased parts, and so on. For more particular remarks the reader is referred to the special part of this work.

We shall here give the remedies and methods in general, which are adapted to the above indications, and shall attempt to make their mode of action intelligible upon scientific principles. This attempt is made rather in the hope of stimulating exact study than as an exhaustive presentation ; such a presentation would be forbidden by the limits of the present work.

We shall first devote a section to the very important group of *external* or *physical remedies*, then speak of the very scanty list of *internal remedies*; in a third section a series of *symptomatic remedies* and methods will be mentioned, which may be employed according to occasion in any spinal disease; and in the fourth section the *general regimen and diet*.

1. *Physical Remedies.—External Remedies.*

Cold.

The application of cold to living tissues first depresses their temperature, and lessens the supply of blood by ischæmia; this retards the processes of metamorphosis, and limits those of exudation and emigration; at the same time the excitability and conductibility of the nervous apparatus are depressed.

Hence are derived the leading indications for the use of cold in inflammations, hyperæmias, and exudations, and also in cases where there is abnormal excitement in the nervous system, pains, and spasm.

In accordance with the latest researches of Riegel[1] and F. Schultze,[2] it can hardly be doubted that the cord can be reached directly by the action of cold, although the thickness of the parts overlying it is so great that a very powerful and continuous application of ice along the line of the spine is necessary to produce that effect. The effects of Chapman's well-known vaso-motor therapeutics are also in favor of the possibility of such an action.

Chapman,[3] by the application of ice or of warmth to the spine, produces a powerful action upon the cord and its vessels. He states that the continuous application of ice produces ischæmia of the cord, lessening the reflex irritability and the other functions; alternate application of ice and warmth increases the flow of blood and the manifestation of energy upon the part of the cord; repeated brief applications of ice at longer intervals produces similar but less marked effects; and, finally, by application of ice to the back we can increase the circulation in those peripheral regions which receive their vaso-motor nerves from the portion of the cord thus treated.

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[1] Virchow's Archiv. Bd. 59. Heft 1.

[2] Locale Einwirkung des Eises auf den thier. Organismus. Deutsch. Arch. klin. Med. XIII. p. 500. 1874.

[3] Med. Times and Gaz. July 18, 1863.

Besides this direct action, a *reflex* influence may be exerted by the cutaneous nerves, as excited or depressed by the stimulus of cold; this has not yet been carefully studied.

The application of cold to the spinal cord may be made either by a common ice-bag or by several, if required, or (better) by Chapman's bags; the complicated apparatus of Koopman[1] does not seem to be required. Less energetic and more transitory effects can be obtained by cold irrigation or cold affusion of the back.

Warmth.

The effects of warmth are in many respects the opposite to those of cold; it raises the temperature of the tissues, increases the flow of blood towards them, and increases the excitability of nervous apparatus. We therefore expect from its action an increased transformation of tissue, or stimulation of the processes of nutrition, and a consequent repair of defects of nutrition, removal of atrophy, degeneration, sclerosis, etc. It is believed to be an excellent remedy for increasing the process of resorption of fluid or solid material, and for repairing chronic processes of inflammation. It furthermore often soothes pain and spasms.

The way in which warmth acts upon the cord has not been much studied. It is even doubtful whether it penetrates directly to the cord when applied externally—though it probably does so. On the other hand, the reflex action, effected by means of the cutaneous nerves, is certainly not to be underestimated.

The results to be expected from its use are: dilatation of the blood-vessels, an increase in the quantity of the current of blood and the other fluids, and of the processes of tissue-change in the cord, an increased facility and rapidity in the nervous processes, and finally, a removal of excitements which often affect the cord, originating in the cutaneous nerves.

It follows from this, in what morbid conditions of the cord the application of warmth will be considered most desirable. But let it not be forgotten that warmth is known to be easily capable of producing over-excitement and exhaustion, leading to congestive states of the cord, and that the use of warmth is contra-indicated in all cases where such results are to be dreaded.

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[1] Berliner klin. Wochenschr. 1870. No. 48.

The methods of applying warmth are very simple: cataplasms, hot sand-bags, Chapman's caoutchouc bags filled with hot water and laid along the spine; hot-water fomentations, or (the mildest form of all) Priessnitz's wet wraps, which gradually warm themselves.

Baths.

Compare *Braun*, Balneotherapie. 3. Aufl. 1873.—*Valentiner*, Handb. der Balneotherapie. 1873.—*Seegen*, Heilquellenlehre. 2. Aufl. 1862.—*Helfft-Thilenius*, Handb. der Balneotherapie. 8. Aufl. 1874.—*Durand-Fardel*, De la valeur des eaux minérales dans le traitement des paraplégies. Bull. de Thérap. Mai 30. 1857. — *Gotth. Scholz*, Ueber Rückenmarkslähmung und ihre Behandlung durch Cudowa. Liegnitz. 1872.—*Runge*, Die Bedeutung der Wassercuren in chronischen Krankheiten. Arch. f. klin. Med. XII. p. 207. 1873.—*Fr. Richter*, Ueber Temperatur und Mechanik der Badeformen bei Tabes und chron. Myelitis. Deutsch. Zeitschr. f. prakt. Med. 1875.

Baths form a very important group of remedies in complaints of the spinal cord. Great results must be ascribed to them, in most of the chronic forms. It is very difficult, however, to define their mode of action and their indications, partly on account of our defective knowledge of spinal pathology, partly because the subject of balneotherapeutics has as yet received but little scientific development. The diagnosis of spinal disease is certainly in a very defective condition; our ideas regarding the anatomical changes, as existing in individual cases and at determined periods, are equally imperfect; hence the uncertainty in regard to indications, and the wide room for empirical treatment, which too often passes beyond the allowed limit of experiment.

We will here speak of the different forms of baths, and their mode of action as taught by the science of balneotherapeutics, and shall attempt to state which forms are most suitable for the treatment of different spinal diseases or groups of symptoms.

Warm Baths

have from time immemorial been a favorite method of treating diseases of the spinal cord—especially paralyses, which usually form the chief part of such as come for balneological treatment. This has been especially true of the indifferent or acratothermæ; but the weak brine baths, alkaline waters, sulphur-baths, etc., containing unimportant amounts of salt and gas, are quite similar to their mode of action. The same is true of steam-baths, hot sand-baths, and the like.

The *effect* of warm baths is first seen upon the skin, in which a great dilatation of the cutaneous vessels occurs, followed later by a moderate contraction. Thereby the circulation in the skin is hastened, and a great evaporation and sweating follows, which is rendered easier by the removal of the upper layers of epidermis. At the same time, the organism receives an access of warmth, or, at least, is enabled to lay up a part of its own store of heat. Thus the processes of oxidation are furthered, and the discharge of most of the functions of the body is facilitated, which accounts for the refreshing effect of a warm bath when one is greatly fatigued.

For our purpose, the effects may be summarized as follows: The warm bath increases the facility of all the chemico-physical processes in the system, leading to stimulation of the metamorphosis of tissue and freedom of function, without any subsequent reaction from excessive stimulation. At the same time, by sheltering the body from the continual change of temperature of the outer air, it acts as a sedative. By the fluxion of blood to the skin it changes the distribution of this fluid, and acts as a derivative in congestions of internal organs; it acts as a resorbent by stimulating the nerve-centres, by changing the course of the blood, and by the production of sweat, and by washing out the system.

Warm baths are best borne by feeble individuals, whose power of resistance and of making heat are weakened. Their effect depends very greatly upon their temperature. If this be indifferent (32°–36° C. [90° to 97° Fahr.]), they are believed to act chiefly as sedatives; warm and very warm baths (36°–42° C. [97° to 108° Fahr.]) are more stimulating, produce strong excitement of the blood-vessels, much sweating, increase of tissue-changes. Lukewarm baths (28°–32° C. [83° to 90° Fahr.]) are believed to have a special depriment effect in the case of nervous, irritable persons.

As the temperature rises, therefore, the exciting qualities become more prominent; as it sinks, the soothing.

The geographical site of the baths must be considered, together with the temperature, especially in the case of indifferent thermæ, for experience seems to show that the higher the site the higher may be the temperature that is borne, and that, the more irritable the patient is, the more elevated may be the spot to which he is sent for cure. This is an important point in practice.

The *indications* which follow from these principles are not easy to state. Most of the circumstances are very complicated. If we confine our attention to the most prominent symptoms, the matter seems very easy; when the symptoms of irritation preponderate, and irritability is very marked (spinal irritation), we choose rather the soothing baths; if symptoms of depression are prominent (anæsthesia, paralysis, etc.), the exciting baths at a higher temperature.

But unless we consider that a decided degree of irritable weakness not only may be, but usually is present in cases of spinal paralytic affection, and that in such cases we usually have to do with an extremely irritable and exhausted nervous system; unless we bear in mind that important disturbances of circulation and nutrition are usually present in the most important organs, which may possibly be affected injuriously by the warm baths, it will be impossible to avoid false steps. Nor have they been avoided, in fact; certain spinal diseases (tabes, myelitis, etc.) have often been made worse by too warm baths.

The degenerative and sclerotic forms of spinal disease seem to require the greatest care in this respect, and we are yet in need, before we can be quite safe in using them, of a more careful study of the special action of thermæ upon these disturbances of nutrition.

Our remedy, therefore, has not merely a directly exciting or composing influence upon the nervous system, but also one of far greater consequence, namely, the alterative action which it exercises (by virtue of increasing the metamorphosis of tissue and changing the direction of the blood-current) upon coarse and fine disturbances of nutrition. We shall not be able to define the indications until the latter mode of action is better understood.

The warm springs are now used in exhaustion of the spinal cord after typhoid and other severe diseases, or excesses of any kind; in spinal irritation (moderately warm baths); in paraplegia from shock to the cord (energetic use of very warm baths); in tabes (avoid very warm baths! select baths of indifferent temperature); in myelitis and softening of the cord (slightly warm); in meningitis exsudativa (all warm springs, especially those of higher temperature), etc.

Fr. Richter believes that only baths of a moderate warmth or moderate coolness ought to be used in chronic inflammatory or atrophic affections of the cord. The warmer baths (from 32.5° C. [90° Fahr.] upwards) are considered by him the best adapted for chronic inflammatory states of the cord with preponderant symptoms of irritation.

We give the following list of the most frequented warm springs, with the height above the sea-level and the temperature of the water: Schlangenbad (900′; 30–32.5° C. [86° to 90° Fahr.]); Badenweiler (1,425′; 30–32.5°); Landeck (1,398′; 31.0–32.5° [88° to 90° Fahr.]); Wildbad (1,323′; 35.0° [95° Fahr.]); Ragatz (1,570′; 38.0° [100.5° Fahr.]); Pfeffers (2,115′; 38.0°); Römerbad (755′; 38.0°); Gastein (3,315′; 32.5–40.0° [90° to 104° Fahr.]); Warmbrunn (1,100′; 40.5° [105° Fahr.]); Wiesbaden (323′; 34.0–40.0° [94° to 104° Fahr.]); Teplitz (648′; 37.5–42.5° [99° to 108° Fahr.]); Leuk (3,309′; 39.0–50.0° [102° to 122° Fahr.]); Baden-Baden (616′; 46.0–68.0° [115° to 154° Fahr.]); Plombières (1,310′; 19.0–62.0° [66° to 144° Fahr.]). A proper selection may be made by attending to the special indications, the individual conditions, etc.

Weak brine-baths (containing not more than one per cent. of chlorides), most sulphur-baths, and the weak alkaline springs act exactly like indifferent springs, and may, according to circumstances, be used in their place.

Steam-baths, *hot sand baths*, *hot air-baths* are of a very high temperature; they produce powerful stimulation and diaphoresis, and may be of use, especially from the latter peculiarity, in very torpid cases of meningitis exsudativa. But it is always necessary to exercise the greatest caution in employing them for spinal complaints.

The so-called *Scotch douche* (alternation of hot and cold water) produces a very exciting effect upon the skin and the nervous system; it has also been recommended in spinal paralysis; it requires to be used with great caution.

Brine-Baths

are very like warm baths in their effects; the action of temperature is the same in both cases, to which the effect of the salt contained must be added. The most suitable proportion is between two and four per cent. The special effect of this ingredient appears in a powerful stimulus of the nutrition and circulation of the skin, increase of the change of tissue, repair of disturbed nutrition, increase of resorption. On account of the exciting effect of the salt, their temperature may be somewhat lower than that of the plain warm baths. Their indications in spinal disease are the same as those for warm baths; and besides, they are often prescribed for the causal indication, in treating scrofula, vertebral disease, caries, etc.

The air of the salt-pans, which may be breathed in many places while the patient is taking his bath, is cool, refreshing, ozonized, and in the case of many irritable patients is much to be desired as an accessory.

The *warm brine-baths containing gas* are far more important than the simple warm ones. This class is represented by Rehme-Oeynhausen, Nauheim, the Schönbornsprudel in Kissingen, and the Soolsprudel in Soden a. T. Their effect is due to their temperature and the salt they contain, in the first place, and, next, to the abundance of carbonic acid, which acts as a powerful excitant of the skin and the nervous system. Its direct effect is a moderate subtraction of heat, followed immediately by a reaction, during which the withdrawal of heat continues, and at the same time a continuous stimulation of the nervous centres. It is as it were a combination of the stimulating and calming effects of the cool and the warm bath.

The result is a general improvement of nutrition and the organic functions; hence, resorption and removal of pathological products; at the same time, excitement of the nervous system may improve the nutrition of that department.

These baths are cool; they must not exceed 32°C. [90° Fahr.]; they are usually taken without moving the water, but if we wish to increase the effect, the water may be agitated.

They are indicated in weakness of the spinal cord following difficult convalescence or other exhausting influences, in tabes, in paralysis after meningitis, in myelitis, spinal paralysis of children, spinal irritation, etc.

Chalybeate Baths,

so-called, are baths with a very slight amount of iron, a more or less considerable amount of salt, and a very considerable quantity of carbonic acid.

Their action is usually referred by balneologists to their temperature and the carbonic acid they contain, while the amount of iron is believed to be insignificant. The physicians continue to swear by the springs, but they fail to make a probable argument for the effects of the iron, except as used internally.

It is certain that these baths, owing to the presence of carbonic acid, are among the most powerful stimulants of all, if warmed with precaution, so as to retain as much of the gas as possible.

They are indicated wherever the thermal brine baths with much gas are required; they should be avoided in all conditions in which great excitation is to be feared; but they should be applied wherever we have to do with a torpid, inexcitable state of the nervous system, especially when anæmia is likewise present.

While in general the use of steel-baths abounding in Co_2 is held unadvisable in diseases of the cord, and they are admitted only in very special forms of functional disturbance, Scholz has recently attempted to save the credit of the steel-baths, especially those of Cudowa, in spinal cases, and has stated their indications and results with exactness. He recommends them very highly for chronic congestion of the cord, especially in anæmic persons and the subjects of nervous exhaustion; only by exception in spinal meningitis, limiting their use to torpid individuals, and cases of a sluggish nature; also in the initial stages of chronic myelitis, with hope of success proportional to the weakness and anæmia of the individual and the sluggishness of the case; in the "primary" form of tabes, without inflammatory symptoms, under the same conditions; finally, in all cases, for the sequelæ of spinal concussion, as soon as the stage of excitement is past. The best results are obtained with mitigated baths (diluted with fresh water).

Although Scholz's presentation is not wholly convincing, yet it is proved that steel-baths, if used with care, are capable of doing good service in many even severe cases. They deserve to be tried thoroughly.

Among waters of this class the following deserve mention: Schwalbach (900′), Pyrmont (400′), St. Moritz (4500′), Brückenau (915′), Driburg (633′), Franzensbad (1300′), Cudowa (1235′), the Kniebisbäder (1200–1900′), etc.

Moor-Baths and Mud-Baths,

so-called, form a separate class. Their action is as yet far from being explained; it cannot yet be stated with definiteness. A part of the effect is due to their quality as warm baths, but this is accomplished in some entirely specific and not yet understood way, since they are much less exciting than warm springs. They seem indicated wherever the thermal treatment is desirable, but its stimulant action is not likely to be well borne; especially, therefore, in weakly, irritable, and anæmic constitutions. Especial benefit has been observed in spinal irritation and the so-called tabes dolorosa, then in paraplegias and contractures consequent upon myelitis, lateral sclerosis, compression of the spinal cord, etc.

The temperature and duration must be regulated according to individual peculiarities.

Good moor-baths are found in Franzensbad, Marienbad, Teplitz, Driburg, Brückenau, Meinberg, Elster, Eilsen, Nenndorf, Liebwerda, Pyrmont, Reinerz, etc.

Pine-needle baths, which have been much used and praised, are only warm baths, in which a powerful stimulus is applied to the skin, not by high temperatures or by Co_2, but by the ethereal oil and the extract of pine twigs. They may be used wherever the more stimulating forms of thermal treatment are indicated.

Among the most important agencies that we possess are the cold and cool baths, including the use of cold water in the greatest variety of forms—what is commonly known as

The Cold-water Treatment.

This treatment, having of late years been administered in a rational manner and closely studied, has much increased in popularity. Its results in all possible forms of chronic nervous troubles are extraordinarily good.

There is as yet no entire agreement in respect to the theory and the mode of action of the cold-water treatment. The conditions are very complicated, and it has naturally happened that individual practitioners of the method have reached more or less one-sided views; some consider the exciting or depressing effects upon the nervous system the principal thing, while the effect in modifying the change of material is subordinate; others try to refer all the effects to the vaso-motor action upon the skin, while a third party sees an explanation of all the leading phenomena in the alterative action upon the metamorphosis of tissue.

It is certain that in the cold-water treatment we observe on the one hand an action upon the cutaneous nerves, and thence transferred to the entire nervous system; also, effects upon the cutaneous blood-vessels, and through them upon the distribution of the blood throughout the circulation; and finally, changes in the circulation and the entire process of transformation of tissue, to which must be ascribed a very special influence upon the cure of severe chronic diseases.

Our present knowledge of the subject amounts to about the following:

A *direct action upon the nervous system*, of an *exciting* or a *depressing* nature, can be produced by the application of cold water; the subtraction of warmth acts as a depressant, the stimulus of the cold as an excitant. According to the form, the temperature, the duration of the baths, we can make the one or the other effect more prominent (Petri).

The *soothing* effect is always produced where the same portion of water remains in constant contact with the skin, as in half, full, and sitz baths without motion; in the wet pack, in wet rubbing without moving the cloth.

The *exciting* effect is always produced when the layer of water in contact with the body is continually changed, so that the stimulus of cold is constantly renewed; that is, in half, full, and sitz-baths with agitation, in rubbing down with a cloth which is moved, in washing, affusion, shower-baths, douches, surf-baths, sea-baths.

The lower the temperature of the water, the more quickly and forcibly do these effects appear. The exciting action of the baths may be increased by previously wrapping the patient in dry blankets, while at the same time an excessive loss of heat is prevented, as the quantity lost in the bath is no greater than what has been saved by the wraps; this method is therefore of special value for persons who need to save their strength. A frequent repetition of these excitations increases the energy of the nervous system.

In regard to the *circulation*, the following effects are to be seen in the *skin*, due for the most part to nervous influence: great ischæmia, goose-flesh, shivering, and soon (though at various intervals), dilatation of the vessels, increased amount of blood, increased secretion from the skin and perspiration, increased development of warmth. These are the symptoms of *reaction*, which occurs with different degrees of facility in different persons, and is of very great importance in the cold-water treatment. In order to its proper development, a certain measure of force, a certain resistance is required; in badly nourished, weak, irritable, and anæmic persons, or those with degenerative disease of important organs, it occurs much less readily; for this reason these persons do not bear the cold-water treatment.

The reaction is lively and vigorous in proportion to the lowness of the temperature of the water and its degree of motion, that is, the force of the stimulus. It is favored by rubbing the skin at the same time, and especially by energetic mechanical dry-rubbing after the application of the cold.

A frequent repetition of these actions upon the skin *increases the circulation and nutrition of the skin permanently*, thereby producing a *change in the distribution of the blood.* A more important effect is its derivative action upon chronic congestions of internal organs, especially the spinal cord. But this can only be obtained with safety when at the same time all injurious irritation of the diseased organ is avoided.

The cold water may act *more directly upon the circulation in internal* organs, by producing ischæmia of these organs through reflex action; thus, Runge states that quite cold baths, applied to the lower extremities, have a direct vaso-motor action upon the cord, diminishing the quantity of blood contained in it. But in aiming at this, it is important that the reaction in the skin should have developed before that in the spinal vessels is commenced, in order that the secondary hyperæmia may be directed principally to the skin. For this purpose it is useful to apply water not merely to the whole surface, but especially to the parts which are related more closely to the affected organ, that is, the skin of the lower extremities and the back.

In regard to the processes of nutrition and change of tissue, it is settled beyond a doubt that every subtraction of warmth from the outer skin is followed by a considerable increase in the production of warmth (this forms a portion of the phenomena of reaction); that the secretions increase, the appetite is improved, etc. It is further probable that the repeated excitation of the nervous system has directly an influence upon the changes of tissue, increasing the formation and destruction of most of the tissues; and that the nutrition of the portions of the central nervous system which are affected by the stimulus is stimulated and improved. We are inclined to believe that this method will certainly cure slight disturbances of the nutrition of the cord, and that, under some circumstances, even severe degrees of change may be gradually repaired.

In brief, then, cold water acts as follows: It strengthens the functions, improves the nutrition, increases the circulation of blood, in the skin; it thus alters the distribution of the blood, and the process of circulation in the system; it relieves, at first temporarily, afterwards permanently, hyperæmia of internal parts (F. Richter); it quiets or excites the nervous system in various degrees; it tones the nervous system by the functional excitement and by the improved nutrition; it accelerates the process of change of tissue, and increases the total nutrition; it promotes resorption and formation.

If we add to this the effects which may be had from certain forms of baths, the increased sweating, the consequences of the increased consumption of water, the muscular movements which are necessarily increased, the effects of diet, climate, altitude in the case of cold-water cures, it becomes evident that we possess but few remedies which have so powerful and various an influence upon the nervous system.

In point of fact, the cold-water cure has been much and advantageously employed in diseases of the cord. Thus, for conditions of irritable weakness of the cord (chiefly by withdrawing caloric and moderate stimulation—cold wraps kept on till warm; rubbing down with moderate friction), for passive hyperæmia of the cord (washing and affusion of the back, stimulant sitz-baths, and long-continued moist cold wraps applied to the body), for fluxionary hyperæmia (soothing friction, soothing sitz-baths with cold compresses on the back, etc.), for tabes dorsalis (chiefly mild treatment, according to circumstances more stimulating or soothing), for chronic myelitis (the same).

The greatest attention must always be paid to the individual; it should not be forgotten that every powerful action of cold produces a powerful reaction, to sustain which a certain amount of force is needed; for which reason, let none be subjected to the treatment but those who possess a certain power of resistance. Weakly, irritable, anæmic persons bear only the more soothing or the very mildly exciting procedures, under certain precautions. In all cases, it may be set down as a rule that the temperature should not be below 20° C. [68° Fahr.], unless special trial shows that lower temperatures are well borne.

The Sea-Bath

is a special and very important form of the cold-water treatment. Its effects are very energetic, because several factors coincide, among which the sea-air is by far the most important. It is, properly speaking, a climate cure associated with a very powerfully stimulant form of the cold-water treatment (*i. e.*, a full bath in vigorous motion, at a very low temperature). The proportion of salt in the water of the North Sea, the Mediterranean, and the Atlantic is equivalent to that in a brine bath of medium strength, and serves to increase the action upon the skin.

The result is a great increase in the change of tissue, increased excretion and formation, increased need of nourishment, increase of bodily weight, tonic action upon the nervous system.

But the sea-bath is adapted only to able-bodied persons, whose appetite and digestion keep pace with the requirements made upon the system. Weak persons with feeble appetites or bad stomachs are not suited; the most that can be done for them is to cause them to enjoy the sea-air, or, in some cases, to add thereto the benefits of bathing in warmed sea-water.

The very powerful stimulant and refrigerant effects of sea-baths make them unfit for most cases of spinal disease. They are useful in spinal irritation and spinal weakness, but only in able-bodied persons; in tabes and similar diseases, only in the lightest form and at the very beginning, or as a concluding treatment after cure is nearly complete—but only when the organs of assimilation are in a fairly good condition. In any case, the *baths* must be used with great care, and the chief value ascribed to the breathing of sea-air, to the climate cure.

Climate Cures

There exist no spinal climate cures for disease of the spinal cord. But it is known that a very favorable influence is exercised upon many severe neuroses, including those of spinal origin, especially when of a functional nature, by certain climates and regions.

The *sea-air*, of which we have spoken, is an example of this. Very much the same is true of the mountain air; it has the same power of stimulating the changes of tissue and the appetite, it aids the performance of sensory and motor functions; the bodily exercise taken in the mountains has an especially enlivening effect upon the nervous system. The higher and drier the site, the more marked are these tonic effects—as, for example, in the Ober-Engadin.

Beneke[1] has made comparative examinations of the action of sea and mountain air, and has reached the conclusion that the increase of metamorphosis of tissue is decidedly greater at the sea-shore than on mountain-summits (3,000′-6,000′), because the subtraction of heat is quicker and greater. Irritable, nervous people, who cannot bear great alteration of tissue, had better choose a residence in the mountains. Experience confirms this in a satisfactory manner.

Every one knows the beneficial effects of a continued residence in various elevated places in Switzerland, the Tyrol, etc., upon so many nervous patients, those with spinal irritation, spinal weakness, impotence, and so forth. And in the more severe cases, the same may be recommended as after-treatment.

The choice among suitable places is extremely large: it should be made with careful regard to all individual peculiarities.

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[1] Zur Lehre von der Differenz der Wirkung der Seeluft und der Gebirgsluft. Deutsch. Arch. f. klin. Med. XIII. p. 80. 1874.

After thus enumerating the different forms of baths and their modes of action, let us formulate briefly the indications, giving some theoretical deductions, which may be of use to the beginner as a clew to treatment, though they require much further confirmation by practice.

a. Purely functional disturbances of the cord, fine changes in nutrition without demonstrable alterations—as in spinal irritation, spinal weakness, concussion without coarse lesion, etc. For the treatment of these we may take into consideration warm springs, thermal brine baths abounding in gas, steel-baths abounding in gas, the cold-water cure, sea-bathing, and mountain climates. The selection must depend, firstly, upon the *individuality of the patient;* for irritable, weak persons without much endurance, let the acrato-thermæ be chosen; the more irritable the subject, the higher should be the location. Or at most, a mild cold-water cure with moderate temperatures. For more vigorous persons with good digestion, the cold-water treatment and the sea-bath, or the thermal brine baths, may be considered. For very torpid persons, the same baths and the steel springs. The choice must further depend on the *morbid symptoms* and their personal variation; with violent symptoms of irritation in very irritable patients, the cooler acrato-thermæ, the more soothing forms of the cold-water method, sea-air, a mountain climate; with preponderant symptoms of debility in torpid persons, the warm springs, the stimulant forms of the cold-water treatment, the thermal brine springs, steel-baths, and sea-baths.

b. Hyperæmic states of the cord and its membranes. For passive hyperæmia (so-called hæmorrhoidal tabes, etc.) the stimulant methods of cold-water treatment, steel-baths, and thermal brine baths are the best. The thermæ are contra-indicated. For active hyperæmia, select the more soothing forms of the cold-water treatment, combined with derivation to the skin (soothing frictions and sitz-baths with cold compresses to the back, etc.). Thermal brine baths and steel-baths require great precaution. Warm springs and sea-baths will usually do harm.

c. Chronic inflammation of the spinal membranes, especially if associated with *fluid exudation*. Here warm springs, thermal brine baths, a powerful cold-water treatment will be useful; in short, anything that promotes resorption and vigorously stimulates the processes of metamorphosis of tissue—but always subject to the distinctions and contra-indications already stated under *a*.

d. Chronic textural changes in the cord itself: inflammation, degeneration, softening, atrophy, sclerosis, etc. Little is to be expected except in the earlier stages of these complaints, and in slight cases. The object is, to stimulate moderately the transmutation of tissue, and the cord, so as to remove the disturbance of nutrition; and for this purpose warm springs, thermal brine baths, steel-baths, moor-baths, and cold-water treatment are useful. But we must always be extremely prudent. We should remember that our patients are almost always irritable and weak persons, suffering from a serious organic lesion, incapable of enduring any severe shocks; and that any increase of the hyperæmia of the cord may prove dangerous to them. Therefore, use only warm springs of an indifferent or lukewarm temperature; mild cold-water cures; mitigated steel-baths; well-regulated thermal brine baths. Consider that the effect, at best, must be slow and gradual, and that the cure cannot be completed by a stay of four or six weeks at the baths.

The selection among the different baths should be made according to the peculiarities of the individual; that of the special method, according to the character of the case, the leading symptoms, concomitant hyperæmia, etc. In general, avoid indulging too much hope in these cases!

Such are the general principles for the balneo-therapeutic treatment of disease of the cord. In the special part we shall see how far they may be applicable to the different classes of disease. Progressive experience will, without doubt, greatly modify them.

A great deal might be added under this important head, but we have not the space. It must be constantly remembered that attention to individuals is of the first importance. An exact diagnosis and a thorough estimate of the individual must go hand in hand in forming plans for treatment. And complications, causes, external conditions often have to be considered, so that the selection is extremely difficult. The physician's practical tact and skill have here a wide field for displaying themselves.

Electricity.

Remak, Galvanotherapie. 1858. p. 443 et seq. Application du courant constant au traitement des névroses. Paris. 1865.—*Ranke*, Ueber krampfstillende Wirkung des constanten elektrischen Stroms. Zeitschrift f. Biologie. II. 1866.—*Flies*, Galvanotherap. Mittheilungen. Deutsche Klinik. 1868.—*Erb*, Galvanotherap. Mittheilungen. Arch. f. klin. Med. III. 1867.—*The Same*, Anwendung der Elektricität in der innern Medicin. Volkmann's Sammlung klin. Vortr. No. 46. 1872.—*Brenner*, Untersuchungen und Beobachtungen auf dem Gebiete der Elektrotherapie. Bd. II. 1869. p. 81.—*Uspensky*, Einfluss des const. Stroms auf das R.-M. Centralbl. f. d. med. Wiss. 1869. No. 37.—*Burckhardt*, Ueber die polare Methode. Arch. f. klin. Med. VIII. p. 100. 1870.—*Ziemssen*, Elektricität in der Medicin. 4. Aufl. 1872. pp. 24, 37, and 143.—Also the text-books of electro-therapeutics by *M. Meyer*, *Duchenne*, *Benedikt*, *M. Rosenthal*, *Beard* and *Rockwell*, and others.

No other remedy has, within a recent period, gained so much ground as electricity in the treatment of spinal disease. But few can be compared with it for activity. It is among the numerous services rendered by Remak that he brought spinal disease within the sphere of scientific galvano-therapeutics, and obtained many striking successes with that treatment.

At first, great obstacles stood in the way of a general acceptance of his views ; first, the doubt as to the possibility of reaching the cord with the electric current, which was expressed by no less an authority than Ziemssen ; and then a scepticism in respect to the genuineness of his successes, which was in part justified by the excessive claims of the specialists, and in part was supported by the failures which unavoidably occurred in old, severe, and absolutely incurable cases.

These obstacles are now overcome ; no one now doubts the possibility of reaching the spinal cord with the electric current, and the numerous and unanimous experience of almost all electro-therapeutists no longer allows us the right to doubt that electricity has accomplished many, in some cases very remarkable, cures in diseases of the cord, and that it has essentially improved the hopeless prognosis of many spinal diseases.

We must therefore pay especial attention to this remedy.

In this place we have only to speak of the *direct treatment of the lesion of the cord.* For in most diseases of this sort there are two things to be distinguished: the electrical treatment of the cord itself and its affection, and the treatment of separate symptoms of the affection. Each of these two may be attended to separately, or they may be combined. As to the second point, we refer to the proper section in Volume XI. of this Cyclopædia, in which the electrical treatment of paralyses, anæsthesiæ, neuralgias, spasms, etc., is given in full.

Here we encounter at once the question of the *mode of action upon the cord and its diseases.* Upon this point little is known, and the material in our possession is more than scanty.

Physiology furnishes us with almost no facts to the purpose. The experiments on stimulation of the cord have produced few results of consequence; it is still a debated question whether the substance of the cord is excitable or not, and whether all the phenomena of excitation may not be referred to irritation of the roots. The question, however, seems to have been answered in the affirmative by the studies of Fick, Engelken, and Dittmar, showing that at all events the white columns of the cord are excitable. Some facts, also, discovered by physiologists (Nobili, Matteucci, Ranke), in relation to the power of the galvanic current to quiet spasm—facts discovered during the treatment of toxic tetanus by strong galvanic currents sent lengthwise of the cord—are perhaps useful, but they require a fresh revision, in view of the more recently discovered facts of the action of powerful currents in arresting reflex actions. The primitive researches of Uspensky, which have shown that the cord acts as a peripheral nerve (!), both for conduction and for reflex actions—that it assumes the conditions of anelectrotonus and catelectrotonus—are quite useless for our purpose. Of the action of electrical currents upon the nutrition of the cord, nothing is known by physiology.

Almost all our knowledge is drawn from *pathological and therapeutical experience*, and consists chiefly of purely empirical facts, which are usually far from being understood.

Our positive knowledge is really limited to this: *that a number of diseases of the cord, especially chronic forms, are relieved or cured during the use of electricity*, employed in various ways. The precise connection between the therapeutic results and the methods used is, however, usually obscure, especially as, in many cases, we are unable to form any clear idea of what diseased condition exists in the cord and its membranes, or what it is that we are removing by electricity.

We know, further, that individual symptoms of spinal disease may be removed by electrical treatment of the cord, as tetanic spasms, contractures, tremor, lancinating pains, anæsthesia, paralysis, etc. But these facts are not always beyond a doubt; for example, the results obtained by Mendel in tetanus with galvanic currents may be referred to a simultaneous action upon the peripheral nerves. In the interesting observations by Dr. Rabow, communicated by Leyden,[1] the hypothesis of a simultaneous action upon the periphery is almost always admissible; and the explanation of the facts there described is at the present time impossible.

We possess, therefore, only inferences and hypotheses regarding the possible action of electricity in the various categories of disease.

Thus, in the so-called *functional disturbances* of the cord, we may first consider the exciting and modifying agency of electric currents; probably, however, the so-called catalytic action (comprising effects produced upon the blood-vessels, upon resorption, osmosis, change of tissue, etc.) is of importance, as relieving the minute disturbances of nutrition.

In *disturbances of circulation* (hyperæmia, stasis, exudation) the explanation may be deduced from the powerful action of electricity upon the vessels and the vaso-motor nerves, upon the circulation of fluids, resorption and the like.

In marked *anatomical changes* (chronic inflammation, degeneration, atrophy, etc.) we must again refer to the "catalytic" action of the current, which can alone explain the fact and the rationale of the cure of these diseases.

It follows *that the so-called catalytic action of the electric current probably constitutes the chief point in the treatment of most diseases of the cord.* It is a pity that this action is so obscure, and so little susceptible of an explanation. We do not care to present hypotheses.

The catalytic action is probably quite independent of the direction of the current. The special action of the two poles is also very obscure in this respect, although the attempt has been made to define it. The simple passage of the current, in sufficient strength and for a sufficient time, through the diseased region, seems to be the essential thing. All details of application must be discovered by empirical methods.

From these principles, the chief rules for the electrical treatment of the cord naturally follow.

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[1] Klinik der Rückenmarkskrankheiten. I. p. 185.

The first point to be deduced is *that the galvanic current should be used almost exclusively* in these affections; first, on account of its physical properties, it being able to penetrate more easily and surely to the depth required than the faradic current;[1] and second, on account of its preponderant catalytic action, which is possessed in but a slight degree by the faradic current. Experience has fully confirmed this position; even the most obstinate adherents of the faradic current claim for it little or no success in organic diseases of the cord. We shall see further on, however, that they are possible to a certain extent.

As regards the special *methods* of applying the galvanic current[2] in diseases of the cord, we have first to settle the question, whether the cord can be reached at all.

The possibility of this is proved by my experiments upon the dead subject, and by numerous facts, discovered by myself and other observers in the case of the living human subject; not to mention numerous therapeutic successes. No one now doubts the possibility of reaching the spinal cord with the galvanic current.

The principal object, in most cases, is to produce *as general and as powerful a passage of the current through the spinal cord* as is possible, especially in its diseased portions.

The cord is a deep-seated organ, and we must employ quite powerful currents in order to reach it. And this can be suitably accomplished without hurting the patient, only by using *very large electrodes*, applied not too near together.

Too many offences are committed against this rule, which is a simple inference from Ohm's laws. If small electrodes are used, the strength of current required to affect the cord sufficiently will produce intolerable pain.

I have often seen persons with sores on their backs, produced by the very painful application of Stöhrer carbon electrodes (quite unfit for this purpose); but who have not seemed to me to have derived the least benefit from the treatment. I always use, for galvanization of the cord, electrodes of *at least* ten centimetres (4 in.) long and five (2 in.) broad.

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[1] *Helmholtz*, Verhandl. des naturhistor. med. Vereins zu Heidelberg. Bd. V. p. 14. 1869.

[2] The same principles apply for the most part to the use of faradism.

The greatest density of current is found immediately under the electrodes; the portions of tissue lying between them are not reached by any great quantity of the current, probably not enough to have any therapeutic effect; hence it follows *that the diseased regions should be brought in contact with the poles throughout their whole extent.* According to circumstances, inclination, or theoretic views, the operator will prefer for the purpose the one or the other pole. As the action of the poles upon the cord is unknown, and as probably both poles are useful in producing the catalytic action, it is perhaps desirable in many cases to let both act, one after the other.

The *relative position of the two electrodes* will be determined by the seat and the extent of the lesion in the cord.

If the disease is chiefly distributed in the longitudinal direction—and this is the most usual case—it is probably best to place both poles on the vertebral column, one in the lumbar region, the other at the nucha. One pole, say the lower, being fixed, the other may be moved quite slowly down over the back, and thus brought in contact with a great part of the cord; and likewise, the upper being fixed, the lower may gradually be carried over the most part of the cord. Thus the "stabile" effect may be made to preponderate, as is desirable in producing catalytic actions, while the point of application is gradually changed.

In *diseases of a more circumscribed locality* (apoplectic foci, spinal paralysis of children, circumscript myelitis, etc.), we shall do best to cover the diseased spot entirely with the pole which is intended to produce its effect, and to put the other pole upon the anterior surface of the body, the abdomen or sternum; this is the surest way of causing a direct passage of the current through the cord; or if desired, we may cause both poles to act, one after the other. Here, also, the electrodes should be made as large as possible.

In all these applications, interruption or reversal is to be avoided as far as possible, unless there exist special indications for their employment.

The direction of the current seems to be nearly indifferent, in respect to results. In general—probably from some undefined impression—the ascending current is preferred. The chief point will always be tc secure the action of the single poles upon the entire extent of the cord.

In selecting the poles there are certain rules observed which require to be confirmed by accumulated experience. Thus, we may be decided to prefer the *anode* in case symptoms of irritation are prominent, in excitable and sensitive persons, in recent, active processes, and when we fear evil results from secondary hyperæmia. The action of the *cathode* will be preferred in disease of a more torpid nature, in persons but slightly irritable, in old processes, associated with thickening and increased dryness of the tissues (atrophies, scleroses, etc.). Generally, however, both poles will be used with advantage.

The cord may also be influenced *indirectly* by the electric current.

It may be reached by the so-called indirect catalysis of Remak, through the *sympathetic*. This process is said to enable us to exercise a regulative influence upon the processes of nutrition in the cord, by galvanization of the cervical sympathetic, by excitation of the vaso-motor (and trophic) paths which pass through it. The influence is a possible one, and even probable, but is not proved. Flies has made further observations upon this point. The possibility of this action has induced me in many cases to apply my treatment in such a way that the sympathetic may be included in the action. The cathode is fixed over the ganglion superius on one side of the neck, and the anode upon the opposite side of the spinal column (quite close to the spinous processes), first between the shoulder-blades, then moving downward very slowly (avoiding labile treatment) till the conus terminalis is reached; and then the same is repeated upon the other side. To this is usually added direct treatment through the vertebral column, anode below and cathode above, slowly changing their positions. I believe that I have seen excellent results from this treatment; perhaps, however, this depends merely upon the more favorable way in which the cord is reached by the current.

The cord may also be influenced *from the skin*. A reflex stimulation, transferred from the sensory nerves to the cord, may very well act like a direct stimulation. Certainly a great part of the effects of the cold-water treatment, or of carbonic acid in baths, and the like, depends on this circumstance. We have nothing precise upon this point; it seems probable, however, to me, that a part of the results which are obtained in spinal diseases by peripheral electrization of the skin and the muscles, is referable to this cause. For this purpose cutaneous faradization and faradization of the muscles may be employed, as well as galvanization. These are the cases which give faradization a just title to comparison with galvanism.

Thus, in M. Meyer (3. Aufl. p. 336), there is a case of spinal disease, which was cured by faradization of the skin with a wire brush.

The remarkable centripetal action of the galvanic current, produced by irritation of peripheral nerves, discovered by Remak,[1] was thought by him to promise great results in practice; but the matter has remained in obscurity, not having been further looked into.

The "general faradization and galvanization," recommended by Beard and Rockwell,[2] probably acts like peripheral faradization. It consists of a stimulation of the skin and the muscles, extending over the whole body.

Beard[3] has also recommended a method of "central galvanization" as especially efficacious in all sorts of central lesions, especially in conditions of spinal exhaustion. It may be useful in many cases. The cathode is placed upon the epigastrium, while the anode is applied successively to the vertex, the vertebral column, the cervical sympathetic, in the labile manner.

Ranke (loc. cit.) believes that an improvement of the condition of the muscles, and the consequent increase in the strength of the normal ascending current of the spinal cord, will act favorably upon spinal nervous weakness, etc.

Finally, M. Meyer[4] has very recently pointed out that galvanic treatment of the points of the spinal column, which are painful under pressure (found in many affections of the cord, especially tabes), often succeeds wonderfully. He applies a current of medium strength for five or ten minutes, placing the anode (unmoved) upon the sensitive points.

The *duration* of single applications may be quite brief—from a minute and a half to five minutes. This is usually enough. Too long applications easily produce disagreeable excitement. The sessions are given daily or less frequently. The duration of the entire treatment is very undetermined; it depends upon the character of the disease and the success obtained. It is often well, after an electric treatment of several weeks, to interpose a considerable period of rest, which may properly be filled up by a visit to spas, a change of climate, or some similar alternative. The diseases are usually very tedious in their course.

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[1] Allg. med. Centralzeitung. 1860. No. 69.

[2] Med. and Surg. Uses of Electricity. 1871. p. 186, etc.

[3] See *Virchow-Hirsch's* Jahresbericht für 1871. I. p. 376, and 1872. I. p. 404.

[4] Berl. klin. Wochenschr. 1875. No. 51.

It is well not to make too light of the administration of electricity; it is not for every one to undertake. It requires much practice and experience. Manual skill, great technical experience, attention to a great variety of points, careful attention to the individual symptoms are absolute requisites.

With the direct treatment of spinal diseases we often have to associate a *treatment of the symptoms*—as paralysis and anæsthesia, spasm and neuralgia, weakness of the genito-urinary organs, paralysis of sphincters or of the muscles of the eye, atrophy of the optic and auditory nerves, etc. All this must be done in accordance with the general rules of electro-therapeutics, with special regard to the individual and to the seat of lesion.

Blood-Letting

may be indicated, under certain circumstances, just as in diseases of other organs; thus, in severe acute inflammations, hyperæmias and stases, with the consequent irritation or paralysis.

General blood-letting is seldom indicated, and is to be governed entirely by general therapeutic principles.

Local blood-letting is best practised on the back, at both sides of the vertebral column, owing to the connection between the inner and the outer plexus of vertebral veins; wet cups or leeches may be applied there. For many cases of abdominal plethora, hemorrhoidal affections, etc., blood-letting at the anus is very useful.

Derivatives.

These formerly played a very prominent part; scarcely a patient with disease of the cord escaped, even from the severest of them all. The back of a chronic spinal patient was usually covered with the scars of blisters, issues, moxa, and the hot iron. In our day we are much less disposed to use these remedies, and perhaps make too little use of them.

Their application and their modes of action are very manifold, comprising derivative influence upon states of sensory excitement, change in the molecular condition of the nervous system, arrest of reflex actions, withdrawal of blood by the action upon the circulation, derivation of inflammation and exudation by establishing an exudative or purulent inflammation of the skin.

All this may also act upon the spinal cord, and probably does act upon it directly, for the cord is undoubtedly the first of the central organs to receive this class of impressions. But, unfortunately, we know no more regarding it. The statements of Busch[1] in respect to the application of the hot iron in various neuroses, especially spinal diseases, are very interesting. He found at the autopsy of one such a case that the iron, applied to the back of the neck, acts to a great depth; in the deepest layers of muscle in that region there were found portions suffused with blood, and even the meninges under the burnt places were found reddened with hyperæmia. Busch is in the habit of burning on both sides of the vertebral spines, in longitudinal strips; he considers this a very powerful derivant in affections of the cord.

If it is allowable to transfer to the pia mater of the cord the facts observed by Schueller in the pia of the brain — and there can be no doubt of its propriety — then the effect of *very large* sinapisms, or, more properly, vesicants, would be a temporary dilatation, followed by a very marked and continued contraction of the vessels of the pia.

We may employ as derivatives cutaneous faradization, sinapisms, vesicants, pustulating ointments, issues, moxæ, and heated iron. The place for application will almost always be the back, opposite to the diseased spot.

External Frictions.

These are much used and highly esteemed among the non-medical public, but are usually rejected by physicians. In this respect medical scepticism often goes too far.

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[1] Berl. klin. Wochenschr. 1873. Nos. 37–39. Session of the niederrhein. Gesellsch. at Bonn.

It is certainly conceivable that the irritation produced by friction with spirituous or other substances upon the skin may act like the stimulation of baths or electricity, exciting and enlivening the action of the spinal cord and bringing to pass a better functional condition and nutrition in it, or else, that the quieting, soothing effect upon the peripheral cutaneous nerves produced by inunction with warm oil or narcotic salves, or fomentations and wraps of a similar nature, has a soothing action upon the central nervous system, and thus contributes to the removal of diseased conditions. I believe that I have in a few cases, quite accidentally, proved to myself the efficacy of such procedures, and am therefore unwilling to see them quite abandoned.

No great results will be reached, it is true, but the external applications may be used as subsidiary remedies, and to sustain the patient's courage.

According to circumstances, we have the choice between the more soothing frictions (with warm oil, infused oil of hyoscyamus, opium or belladonna ointment, etc.), and the more stimulating and invigorating frictions (with French brandy, spirit of ants, of wild thyme or of camphor, liniment of ammonia or of camphor, etc.).

2. *Chemical Remedies—Internal Remedies.*

Cf. *Nothnagel*, Handbuch der Arzneimittellehre. 1870.—*Husemann*, Handbuch der gesammten Arzneimittellehre. II. 1875.—*Schueller*, Ueber die Einwirkung einiger Arzneimittel auf die Hirngefässe. Berl. klin. Wochenschrift. 1874. Nos. 25 and 26.—*Brown-Séquard*, Lect. on the Diagnosis and Treatment of the Principal Forms of Paralysis of the Lower Extremities. London, 1861. p. 110.

We have entered upon a region which is still very obscure, and in need of thorough study. Of its contents, we know next to nothing; the little that we have learnt from therapeutic experience is neither sufficiently established by facts nor based upon intelligible scientific principles.

We are acquainted with the specific action of some few remedies upon certain functions of the cord, but these actions are seldom required. We are still quite ignorant in regard to most of the remedies we employ, *how* they act upon the cord and its nutrition — although we are especially fond of using them in organic diseases of this organ.

We therefore limit ourselves to as brief an enumeration as possible of the internal remedies, leaving the most of what is to be said to the special part of this work.

A leading remedy in disease of the spinal cord is certainly *strychnia*, with the preparations of nux vomica. Its physiological action consists in a very great augmentation of the spinal reflex action, probably due to a direct irritation of the central ganglion cells by the poison. It also stimulates the vaso-motor centres very powerfully. Upon the motor nerves it seems to have no influence, but upon the sensory apparatus it acts by increasing their capacity for excitement. Finally, it is said to increase considerably the flow of blood to the spinal cord.

Strychnia has been applied in many ways to the treatment of spinal diseases. But in most cases it is totally useless, for the increase of reflex excitability can be of little use to the patient so long as the disturbance of nutrition exists, which causes the paralysis. It then does nothing but produce energetic reflex jerkings in the paralyzed parts, without aiding the restoration of tissues (Gull). In very irritable and easily exhausted patients, in irritative processes in the cord (tabes, myelitis, spinal irritation, etc.) it seems to do positive harm, and its use has been of late almost abandoned. Even such fortunate cases as those lately published by Acker,[1] are hardly to be considered in comparison with the numerous cases of failure.

The use of strychnia is, however, only allowable in old cases of palsy, or when the principal lesion has been repaired without a full recovery of function. (Nevertheless, in the most of such cases electricity will be a much surer and more harmless agent.) It ought also to be tried in purely functional weakness of a torpid nature, in order to add to our knowledge of the subject. In such cases it seems to act in moderate doses as a nerve-tonic. In cases of spinal anæmia producing disturbance of nutrition, it may be used. Finally, it seems to act well in paralysis of sphincters, in weakness of the genito-urinary organs, in enuresis nocturna, perhaps when these disturbances depend on anomalies of the reflex centres in the lumbar cord.

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[1] Arch. f. klin. Med. XIII. p. 438.

The watery extract of nux vomica is given in the dose of from one-half to three grains (0.03–0.20); the alcoholic extract, in the dose of from one-sixth to one grain (0.01–0.06); the tincture of nux vomica, from five to fifteen drops; and nitrate of strychnia, from one twenty-second to one-sixth of a grain (0.003–0.01), best by subcutaneous injection in the same doses.

Coniine acts in some respects in the opposite way. It directly paralyzes the motor nerves, but seems also to have a specific action upon the spinal cord, as it powerfully depresses the reflex excitability. It is therefore available for the relief of spasmodic conditions, especially those of a reflex character. (Dose of the conium herb, from three-quarters to four and a half grains (0.05–0.30); coniine, from one-sixtieth to one twenty-second of a grain (0.001–0.003), dissolved in water.)

Curare has a very similar action, directly paralyzing the motor nerves and diminishing the reflex action of the cord. It s a very uncertain remedy, and may be dispensed with.

Calabar bean lessens and destroys the excitability of the ganglia of the cord, especially in the gray anterior columns, giv-ng rise to paralysis, loss of reflex excitability, and of the sense of pain. This remedy is therefore to be used in cases of exaggerated reflex action (in tetanus, the intoxication of strychnia, reflex contractures). Dose of extract, from one-twelfth to one-third of a grain (0.005–0.02), in solution or pills.

Ergotin (or ergot) has a powerful effect upon the vessels, and according to Brown-Séquard, a special tendency to the vessels of the spinal cord. It is said to produce at the same time a diminution of reflex excitability. It affects all the smooth muscular fibres, especially those of the bladder.

It is employed in fluxionary hyperæmia and paraplegia; in the latter disease it is especially useful (according to Brown-Séquard) when there is hyperæmia or chronic inflammation of the cord and its membranes, while it is contra-indicated in the absence of symptoms of irritation or hyperæmia. (Dose of aqueous extract of ergot, from one and a half to seven and a half grains (0.1–0.5) internally, from one-sixth to one and a half grains (0.01–0.10) subcutaneously; of tincture of ergot, from ten to thirty drops.

Atropine (or *belladonna*) has also a powerful action upon the vessels, and in large doses, according to Brown-Séquard, it specially contracts the vessels of the cord. It further depresses the excitability of the motor and sensory nerves and muscles; its action upon the cord itself is unknown. Brown-Séquard recommends it in the same affections as ergot. (Dose of extract of belladonna, from one-sixth to one and a half grains (0.01–0.10), of sulphate of atropia, from one one-hundred and twentieth to one-thirtieth of a grain (0.0005–0.001–0.002).

Nitrate of silver. This remedy was first recommended by Wunderlich[1] in progressive spinal paralysis, and since then has been much used in tabes and other forms of sclerosis of the cord. It is undeniable that in many cases it has produced most decided effects: that it removes the pain, lessens the anæsthesia, improves the ataxia and the paralysis, and in many cases brings about a complete cure. But its precise mode of action and its special indications are as yet entirely unknown. Injurious effects are reported by many physicians. Dose from one-sixth to one-third of a grain (0.01–0.02), three times a day, best given in the form of pills; to be continued for a long period.

Iodide of potassium has been much used in diseases of the cord, as in so many other neuroses; and not rarely with success. Its mode of action and the special indications for its use are quite as obscure as those of nitrate of silver. Its well-known action upon a variety of pathological products, various forms of inflammation, exudation, etc., furnishes so tempting an excuse, that it is tried over and over again. It is prescribed a good deal in exudative inflammations of the meninges, especially in the chronic stage; in chronic inflammations of the cord itself, especially when they can be ascribed to a rheumatic origin; in new formations, sclerosis, etc. Its chief employment is found, of course, in cases where syphilitic action is suspected. Let the dose not be too small; from fifteen to forty-five grains (1.0 to 3.0) each day. Nearly the same is true of the mercurial preparations, so frequently used.

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[1] Arch. der Heilkunde. II. 1861. p. 193, and IV. 1863. p. 43.

Bromide of potassium, among other effects, lessens the reflex activity of the cord, and afterwards paralyzes the peripheral nerves. It contracts the vessels of the brain, and is believed to act thus as an hypnotic. It therefore appears indicated in cases of increased reflex excitability, and in reflex contractures; it further appears useful in pain, neuralgia of centric origin, sleeplessness, and conditions of sexual irritation. The dose given is from fifteen to thirty grains (1.0–2.0); or from one and a half to two and a half drachms (6.0–10.0 grammes) a day.

A portion of the toxic effects of *arsenic* are produced upon the spinal cord; but we do not know them very precisely. Probably the chief part is due to the effect upon the general nutrition and the tonic action upon the nervous system; it is highly recommended by Isnard, and is given in the usual manner.

Phosphorus has been repeatedly recommended for various neuroses, and in tabes, paraplegia, etc. It has not yet obtained a general recognition; it seems to be very dangerous, and not specially efficacious.

Of the physiological action of *zinc* and its preparations upon the cord and upon spinal symptoms, we know almost nothing. It is much used empirically as a nervine, and may perhaps be of value in conditions of functional weakness. It is most employed in the form of the oxide and valerianate.

Quinia is held by many physicians, both old and young, to be useful to the spinal cord. Physiological researches give us few grounds for this belief. Its use in practice has been confined almost exclusively to its anti-febrile and anti-zymotic effects; it has also an invigorant action. It cannot be doubted that quinia has a powerful action upon the central nervous system, but the manner of its action, and the parts affected, are quite unknown.

It is employed in spinal nervous weakness, in excentric pains, in fever, in spinal complaints which depend on malaria. The dose is graduated according to the effect sought; small doses are the favorite ones for the strengthening effect.

The *sodio-chloride of gold* has been repeatedly recommended as a remedy in spinal complaints; its claim to the title is still doubtful. The dose is from one-sixth (0.01) to three-quarters of a grain (0.05).

We might prolong this enumeration at pleasure, but the reader doubtless has enough; further explorations of this dark and doubtful region will prove of little advantage.

We would add a few words upon the *diet cures*, which are believed to act like courses of internal drug medication. They are not of much importance in diseases of the cord. But milk cures, whey and grape cures, and the like, may produce a desirable effect upon the general nutrition, and thus upon that of the spinal cord.

The same is true of *courses of mineral water*, taken inwardly. Nothing is known of any specific action upon the spinal cord and its affections. Nevertheless, such courses may be required by certain causal or symptomatic indications, and may then have a most excellent effect.

3. *Symptomatic Remedies and Methods.*

We are very often compelled to give *sedatives* in spinal diseases; especially for the relief of the frequent excentric pains, pains in the back, painful reflex contractions and spasms, etc.

In addition to the ordinary narcotics we possess a number of remedies, discovered empirically.

The best of anodynes—opium with its preparations—increases the reflex excitability of the cord, and does not depress it except in large doses. It must, therefore, be avoided in cases of reflex spasm, but is of very great value as a simple antidote to pain. This is especially true of subcutaneous injections of morphia. But the patient must not be allowed to form a habit of relying upon it, as that always exercises an unfavorable influence upon the general progress of the disease, and the habit is extremely hard to break up.

It is recommended to make trial of the remedies above named —coniine, atropia, calabar, etc. It has been found empirically that in many cases bromide of potassium, quinia, etc., have a specially quieting influence.

Electricity also often has a very good effect; the relief to lancinating pains, especially by local faradization or galvanization, is often quite magical—though usually but temporary.

For painful priapism, or great sexual excitement, use bromide of potassium, lupulin, camphor, etc.

For obstinate sleeplessness, the ordinary hypnotics are used; they often fail, and their continued employment is not without danger. We must then have recourse to all auxiliary methods. The cold-water treatment (cold washing of the feet, Priessnitz's packing of the calves) is very advisable.

In other cases we shall make more use of *irritants*, as in paralysis, anæsthesia, weakness of the bladder, etc. Here electricity is the chief remedy. After it, strychnia, ergot, etc.

Tonics are often indicated, as the preparations of iron, the chalybeate springs; also quinia, various bitters and tonics, according to general principles.

In many diseases of the cord, the *treatment of cystitis* forms a specially important point, as this often constitutes the first serious danger to life.

The observance of suitable *prophylactic rules* will be of the first importance in this complaint; and of these the chief one is —never to allow the urine to stagnate in the bladder. The evacuation ought, therefore, to be aided by pressure upon the bladder, or by exciting reflex action by kneading and pressing the wall of that organ; or by raising the patient in order to gain the mechanical advantage. If necessary, catheterize regularly and from the beginning, twice a day, but with extreme precaution and cleanliness. It is also advisable to assist and complete the evacuation by the principle of the siphon, depressing the outer orifice of the catheter below the level of the fundus vesicæ. In all cases we should see that the bladder is frequently evacuated, and should lessen the tendency to decomposition in the urine by making the patient drink copiously of common water, by the use of the waters of Ems, Selters, or Wildung, by exhibition of salicylic or benzoic acid.

If there is incontinence of the bladder, the best means for keeping the patient in a state of tolerable cleanliness consists in a regular artificial evacuation. Frequent washing, the use of portable urinals, or (in the case of women) placing sponges in front, are also necessary.

When catarrh of the bladder has appeared, it may be arrested in its further progress, or even cured, by preventing the process of decomposition, and checking the tendency of the urine to become alkaline. For this purpose *salicylic acid* is found a most excellent remedy, according to the experience of Fuerbringer,[1] which my own in part confirms. From thirty to sixty grains are given daily in watery solution or emulsion by the mouth, and injections into the bladder (1 part to 500) may be added. It brings back the acid reaction, removes the foul odor, and clears up the urine. Benzoic acid is said by Gosselin and Robin[2] to act similarly, and is claimed to be the best agent for restoring the acid reaction to ammoniacal urine, and relieving the catarrh which depends upon this condition. (Dose from thirty to ninety grains daily in emulsion or powders.) The use of lime-water, Wildung water, and various alkaline waters (Ems, Vichy, Selters, etc.), seems to have a good effect in light cases. Clemens[3] recommends ergotin for the same purpose.

As a direct remedy for catarrhal inflammation of the bladder, the usual astringents are given—uva ursi, tannic and gallic acid, copaiba, oil of turpentine, tar-water, etc.

But in all severe cases, accompanied with abundant formation of pus and mucus, a good deal of ammoniacal decomposition, ulceration, etc., it will be indispensable to wash out the bladder, regularly and with great precaution. Injections for this purpose are composed of lukewarm water, which may gradually be made colder, of salt water, weak solutions of tannin, nitrate of silver, salicylic acid (1 : 500), etc. They may be made either with a simple syringe or with the irrigator, but the catheter *à double courant* is the most suitable.

In all such cases it is advantageous, while pursuing the above plan, to attack the palsy and anæsthesia of the bladder with electricity.

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[1] Berl. klin. Wochenschr. 1875. No. 19. Zur Wirkung der Salicylsäure. Jena, 1875, p. 62.

[2] Traitem. de la cystite ammoniac. par l'acide benzoique. Arch. gén. Nov. 1874.

[3] Deutsche Klinik. 1865. No. 27.

A careful regulation of the diet is very important. Patients with decided catarrh of the bladder must use the blandest and most digestible articles of diet, avoiding all acid and highly spiced articles; the use of beer and strong sweet wine is unadvisable; but a light semi-acid white wine, or a good red wine, diluted with water, is allowable.

Of almost more importance than the treatment of cystitis is that of *bed-sores*. These form one of the most horrible complications, infinitely increasing the patient's sufferings, and must be avoided by all possible means, as it is extremely hard to heal them when once fully developed.

The chief remedies are of the *prophylactic* sort, and the principal point to observe is the *avoidance of all continued pressure on the skin*. This can be attained by frequent change of posture, by air-pillows, water-cushions, lying on bags of millet, on buckskin, etc. *All filth and irritation of the skin where pressure is borne must be avoided as far as possible;* removal of fæces and urine, frequent washing, anointing with grease and oil are the chief precautions. And finally, a *slight stimulation of the tone of the cutaneous vessels* may assist in preventing bed-sores, for which purpose frequent washing in cold water, or in spirit, or the alternate application of ice and poultices (Brown-Séquard), or moderate cutaneous faradization may be used.

If a bed-sore appears in a light form (superficial ulceration, furuncles, etc.), it may often be healed by simple treatment, though slowly. Great cleanliness, frequent washing, dressing with zinc ointment, or mildly irritant salves, chamomile water, or aromatic wine will be sufficient in connection with the prophylactic rules.

The matter is less simple in the case of the true gangrenous sore, which often reaches an enormous size, and extends without cessation downwards and at its borders. The first object in this case consists in assisting the throwing off of gangrenous portions, and developing the reactive inflammation; according to Brown-Séquard, the alternation of ice (for ten minutes) with poultices (for one or two hours) is an excellent means to this end. The dead tissues, separated by the line of demarcation, should be got rid of as soon as possible, and an antiseptic dressing applied.

Carbolic acid has great advantages over the remedies formerly used (camphor wine, aromatic wine, "unguentum contra decubitus," etc.). It should be applied in a watery or oily solution, by preference upon good surgical wadding. Under this treatment I have seen the wounds take on the most healthy granulations, and even very advanced bed-sores heal.[1]

It is often very hard to find a suitable position for the patient, especially when there are bed-sores on the sacrum, the ischiatic protuberances, trochanters, heels, etc. Much care and observation must be directed to the prevention of new sores. Water-pillows and air-pillows must be used in their various forms. In the worst cases I have found it useful to swing the lower leg on a pillow, placing the knee-joint and hip-joint in the position of rectangular flexion; this may, to some extent, also free the sacrum from pressure.

4. *General Observations—Method of Life.*

The general care of patients with spinal disease, their diet and manner of life, must be governed strictly in accordance with the requirements of the disease. Many mistakes and negligences are committed in these matters, which neutralize the success of the other treatment.

The rules must differ widely, of course, in different cases.

In the acute, inflammatory, and similar forms, the proper diet, rest in bed, avoidance of all excitement and effort must be ordered.

But in the chronic forms also, in functional as well as organic disturbances, the patient must in general be very careful, and must observe the following general rules (with suitable adaptation to his case), with a strictness proportionate to the weakness and irritability of the patient's nervous system, to the amount of symptoms of irritation present, to the readiness with which he suffers from the effects of external circumstances.

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[1] *Hammond* (Diseases of the Nervous System. 3d edition. 1873), following Crussel and Spencer Wells, recommends a simple galvanic element as an excellent means of curing bed-sores. A thin silver plate is laid on the sore, a similar plate of zinc, with a piece of moist cloth underneath, upon a distant part of the skin, and the two are connected by a wire. After a day or two the beneficial effects are seen. I have no personal experience of this.

The *diet* must be corroborating and tonic in most cases, without being in any way exciting. Milk, meat, eggs, light vegetables, puddings, and fruits are allowed and required; an abundant supply of fatty nutriment (butter, cream, oil, cod-liver oil) is perhaps useful in a good many cases; strong seasoning, and very complicated and heavy dishes must always be avoided. A glass of wine or beer at table is usually allowable, but their excessive use is to be strictly forbidden. Strong tea and coffee must be avoided in most cases. Smoking within moderate bounds is allowed.

In regard to the general *regulation of the life*, the allowable amount of work and rest, of bodily and mental activity, we must be governed chiefly by the state of the patient's strength. It is but rarely that chronic cases can be condemned to continuous confinement in bed; yet there may be many decided indications for it. Brown-Séquard directs the avoidance of lying on the back, as far as possible, in conditions of inflammation and hyperæmia.

It is usual and desirable to allow a moderate amount of active bodily exercise when practicable, but let the patient be warned against every excess of exertion! Great injury is thus often done; for instance, by too long walks; the patient must be especially cautious about this while taking a course at a medicinal spring.

Mental effort, the practice of a profession, cannot always be forbidden on account of circumstances; and the disease is usually so tedious that such a measure would be excessively irksome. Let this, therefore, be allowed within judicious limits, every excess of effort being forbidden, especially night-work, which is especially injurious.

The same is true of sexual indulgence; between different persons and cases there is a very great difference of power. The permission must be determined by the patient's condition; in all cases it is well to limit the indulgence as much as possible, and in many to forbid it wholly; in a small number, moderate indulgence is permissible.

In most cases the need of sleep is among the things to be first attended to, and in connection with this, all exciting and fatiguing society must be forbidden. The patient must also avoid taking cold, and clothe himself accordingly. He may by degrees harden himself by using cold ablutions, and so on.

A great deal of fresh air will almost always be beneficial to patients; let them sit or drive in the open air, especially on the mountain and in the forest. This is the principal reason for ordering many patients to pass the winter in the south, especially when their home is in a rough northern climate, which keeps them in-doors the whole winter. Such patients will find a winter in South-western Germany a great pleasure; others will like the banks of the Lake of Geneva, the Riviera, Meran, Venice, etc., where they may spend several hours every day in the air. All, however, will depend on individual circumstances.

II.

SPECIAL DISEASES.

1. Diseases of the Membranes of the Spinal Cord.

1. *Hyperæmia of the Meninges of the Cord, and of the Cord itself.*

J. P. Frank, De vertebralis columnæ in morbis dignitate. Select. opuscul. med. Ticin. 1792. p. 1.—*Ollivier*, Traité des malad. de la moëlle épin. III. éd. 1837. Tom. II. pp. 1-137.—*Hasse*, Krankh. des Nervensystems. 1855; 2. Aufl. 1869. p. 656.—*Brown-Séquard*, Diagnosis and Treatment of the Principal Forms of Paralysis of the Lower Extremities. London, 1861.—*Hammond*, A Treatise on the Diseases of the Nervous System. 3d ed. 1873. — *Leyden*, Klinik der Rückenmarkskrankheiten. I. 1874. p. 362.—*M. Rosenthal*, Klinik der Nervenkrankheiten. 2. Aufl. 1875. p. 270.—*Gauné*, Epidémie de congestion rhachid. Arch. gén. Janv. 1858. p. 1.—*A. Mayer*, Die Bedeutung des Rückenschmerzes u. s. w. Arch. d. Heilk. I. 1860. p. 373.—*Leudet*, Arch. génér. Mars. 1863. p. 257.—*Desnos*, Observat. de congestion méningo-spinale, etc. Gaz. méd. de Paris. 1870. No. 14. p. 187.—*Steiner*, Fall von Rückenmarkshyperämie. Arch. der Heilk. XI. 1870. p. 233.

It is impossible to treat of hyperæmia of the spinal meninges without including that of the spinal cord. And it is hardly conceivable that any considerable hyperæmia of the meninges should exist without a similar condition existing in the cord also, as the vascular supply of both is the same. The symptoms of meningeal and spinal hyperæmia coincide; their etiology and treatment are the same. Defective as is our knowledge of both forms of disturbance, we must therefore consider them together.

Definition.—By hyperæmia of the cord and its membranes we understand *an increased supply of blood in the tissues contained within the vertebral canal*, the spinal cord, its membranes, and the extra-meningeal cellular tissue. This excess of blood may be due either to an *increased flow of blood*, when it is usually of an arterial character (*active, arterial hyperæmia, fluxion*), or to *arrest of the return current of blood*, when it is usually of a venous character (*passive hyperæmia, venous stasis*). In practice, these two sorts cannot always be clearly separated.

The frequency and importance of spinal hyperæmia have certainly been overestimated hitherto. This results chiefly from the fact, that in many fatal cases those who examined the body have been content with the mere macroscopic appearance of hyperæmia of the cord, without reflecting that a cord which looks quite normal to the naked eye is by no means necessarily so in its finer anatomy. Ollivier, in especial, has gone much too far in this, and has thrown together under the title of "Congestions spinales" a variety of things which are certainly of a much more serious character. The spinal and meningeal hyperæmias certainly need a fresh and more discriminating study.

Etiology and Pathogenesis.

Little is known as to the existence of any special *predisposition* to spinal hyperæmia. On the contrary, it deserves special attention, that the arrangement of the vessels of the cord protects it in a peculiar manner from mechanical disturbances of circulation, as is shown by Hayem's[1] luminous observations upon the distribution of the arterial and venous vessels within the spinal canal. The many anastomosing arteries, coming from all sides, and the enormous venous plexuses, which carry off the blood from the spinal canal both above and below the diaphragm, are the cause of this. The diseases of the vessels of the cord have not yet been accurately studied.

In respect to the *occasional causes*, we must attempt to distinguish the two principal forms of hyperæmia.

Active hyperæmia has certainly some relations to hyperæmia of the brain; this follows from the fact that the spinal arteries originate from the vertebrals. But hyperæmia of the cord usually recedes into the background, as compared with hyperæmia of the brain, if the latter is present.

Hyperæmia of the cord and its membranes is produced—

a. By *functional stimulation* of the cord. In this case, as rywhere, the activity of the organ is accompanied by an in- ased flow of blood towards it; and in case of excessive exer- n the flow may continue and form a pathological state, as in rwork of body, severe marching, violent sexual excitement, ess of coitus, spinal spasm, and so on. The absolute proof the agency of either of these circumstances remains to be ught.

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[1] Des hémorrhagies intrarhachidiennes. Paris. 1872. pp. 7–20.

b. By *nutritive stimulation.* Active hyperæmia accompanies umber of disturbances of the nutrition of the cord and its mbranes, is never absent in acute inflammations of the organ, l in the first stages of the latter is often the only macroscopic pearance.

c. By *toxic stimulation.* Poisoning with strychnia, nitrite amyl, carbonic oxide, chronic poisoning with alcohol or ab- the, etc., produce spinal hyperæmia.

d. By *collateral fluxion*, as in sudden suppression of the nses, in dysmenorrhœa, in the turgor of hæmorrhoids or men- uation, in suppression of hæmorrhoidal bleeding, in sup- ssed perspiration of the feet or when the feet are always cold, . In most of these cases, nothing but the assumption of a sened resistance on the part of the spinal vascular system can plain why this particular region should become the seat of peræmia.

e. By *the effects of cold.* This is thought by Hammond to be most common cause of spinal hyperæmia; a wetting to the n, when one is making too great bodily exertions, is thought ecially dangerous. Cold produces its effects both by collate- fluxion, through interference with the circulation in the skin, l also by a reflex action from the cutaneous nerves upon the nal vessels, which are dilated.

f. By *traumatic causes*, as shock to the cord, a fall on the k or seat, etc. (Leudet); the mechanism of this action is still scure.

g. Finally, severe febrile diseases (typhoid, acute exanthe- ta, malarial infection, etc.) have been observed to be accom- nied by spinal hyperæmia, and the latter has appeared epi- mically in a girls' boarding-school (Gauné).

Of *passive hyperæmia*, the most prominent cause is to be sought in *general venous congestion* or stasis, such as is produced by diseases of the heart and lungs, or accompanies severe spasmodic affections (tetanus, eclampsia, etc.), and occurs during the death-agony; also, in *local venous stasis*, especially such as is produced by obstructions in the portal system, or in the pelvic veins, in the case of hemorrhoids, abdominal plethora, affections of the liver, tumors in the pelvis or by the side of the spine pressing on the venous trunks and plexuses.

Pathological Anatomy.

The anatomical evidence bearing upon hyperæmia in the spinal canal is as uncertain and ambiguous as possible. Comparatively very few spinal canals are opened at autopsies, which makes it hard to estimate the relative proportion of normal and of pathological conditions. The post-mortem changes add a great deal to the difficulty of establishing the fact of hyperæmia; the arteries are emptied, the veins over-filled; we may find, on the one hand, a deceptive disappearance of hyperæmia, which existed during life, and, on the other, a deceptive appearance of hyperæmia, having its origin during the death-struggle or after death, without having existed during life. Add to this the imbibition of the coloring-matter of the blood, and the gravitation of blood to dependent parts—all of them circumstances which may confuse the judgment and obscure the facts.

Nevertheless, the existence of hyperæmia, in marked cases, can generally be detected with certainty.

Active hyperæmia is betrayed by a rose-color or scarlet redness of the cord and its envelopes, by injection of the minute vessels, by tortuosity of the arteries and veins; the white substance of the cord appears of a rose-color; the gray is darkened, reddish-gray, brownish; under the microscope the finer arteries and capillaries are seen crowded with blood. In the more marked cases punctiform extravasations and ecchymoses are seen dotted over the membranes and in the substance of the cord, and often larger extravasations. The spinal fluid is usually increased, is muddy, and of a reddish color.

In *passive hyperæmia* the extra-meningeal plexuses of veins are especially crowded with blood, all their veins are enlarged and tortuous, and the coloration approaches a cyanotic hue. Ecchymoses may be present here also; the spinal fluid is almost always increased to some extent.

In a few cases something is seen which lies between the condition of congestion and that of inflammation.

In chronic or often-repeated hyperæmia the pia and arachnoid are thickened and opaque, and highly pigmented.

These hyperæmias do not always extend over the entire spinal canal, but often are confined to the cervical, or lumbar, or other portions.

The cerebral hyperæmia, and the diseases of other organs which cause spinal hyperæmia, need not here be described.

Symptoms.

In spite of the great certainty with which congestions and stases of the cord are spoken of, their symptoms are still extremely obscure, and the diagnosis is surrounded by many difficulties and doubts. We may therefore sum up the subject very briefly, leaving to the future a more thorough elaboration of this branch of spinal pathology.

The most prominent symptoms are those of *sensory irritation;* the patient complains of *pain in the loins and along the spine;* the pain is dull, oppressive, not very severe, and is not always increased by pressure upon the spinous processes. The additional symptoms of *paræsthesia* (tingling, formication, etc.) and *tearing pain in the extremities* (especially the lower) are soon perceived. A slight *hyperæsthesia of the skin* is usually associated with a *moderate increase of reflex activity.* The girdle sensation is often felt (Hammond). *Symptoms of motor irritation* are more rare; as slight transitory jerking of the muscles, trembling of the limbs, etc. The electrical excitability is said to be often increased (M. Rosenthal).

All these symptoms can be derived without difficulty from the increased flow of blood, and the consequent stimulation of the nervous apparatus, and doubtless belong principally to the condition of active hyperæmia; but they may also, in part, be explained by the mechanical irritation and the violence done to the tissues by the dilated vessels.

Distinct *symptoms of depression* are also seen, and usually make their appearance at the beginning with those of irritation. A *sensation of numbness and heaviness* is perceived in the lower extremities; slight *anæsthesia* is also demonstrable, but seldom in a severe form. *Motor weakness* is never absent, though usually quite moderate in amount (slight fatigue, heaviness of the limbs), and it is rarely—perhaps in simple hyperæmia never—the case that complete paraplegia occurs. Symptoms of weakness or palsy of the bladder seem rare, but are occasionally described. Hammond says that he has often observed erection of the penis.

It is not easy to explain the paretic symptoms. We may take into account the pressure of dilated vessels upon the nervous elements of the cord and the nerve-roots, the pressure exerted by an increased quantity of spinal fluid, and finally, perhaps, the defective nutrition of the nervous apparatus, dependent on the blocking of the circulation.

The symptoms of spinal hyperæmia are, almost without exception, bilateral, and are usually limited to the lower half of the body, or at least they begin in the lower extremities; they seldom ascend to the upper, and if they do the extension is usually rapid; the respiration, in such cases, is said to have been disturbed, with short, dyspnoic breathing, and even paralysis of both facial nerves (Steiner).

Usually the symptoms exhibit a certain transitoriness, which is quite characteristic; they change their seat and degree of severity quickly, and even grave symptoms may completely disappear with striking rapidity.

Brown-Séquard says that he has observed all the symptoms of hyperæmia aggravated by lying on the back, with the head and legs raised, while they were relieved by lying on the face, or by standing and walking, owing to the effect of gravitation upon the circulation in the spinal canal. For this reason such patients are said to feel worse in the morning while in bed. Others say that standing and sitting make the symptoms worse, especially those of paresis, and refer this to the gravitation of the increased quantity of spinal fluid; such patients are better in the horizontal posture.

There is *no fever* in simple hyperæmia of the cord. The *pulse* may be accelerated, or retarded, if the hyperæmia extends to the spinal centres of cardiac innervation. The general health is more or less interfered with.

Distinct groups of symptoms belonging respectively to active and passive hyperæmia have not yet been made out. But conclusions drawn from the preponderant nature of the symptoms will in most cases be correct. If those of an irritative character prevail, we shall think rather of active hyperæmia; if symptoms of depression, of a passive form. But we should remember that most of the symptoms may occur in either form, though in various degrees of intensity.

Course.—The development of hyperæmia of the cord is either sudden, so that the entire series of symptoms is soon completed, or it is slow, and gradually increases in intensity; this is the more usual case. Having been developed, they continue with various degrees of fluctuation for days, for weeks, or months.

The disease usually ends in *recovery;* this is often brought about rapidly, by critical hemorrhages (menstruation, hemorrhoidal bleeding) or therapeutic measures. Relapses are not rare; and the cure is often accomplished by a very gradual disappearance of all the symptoms.

In many patients (those with piles, etc.), we find hyperæmia of the cord becoming *habitual*, returning regularly and frequently. This may lead by degrees to severer disturbances, by the development of chronic inflammations and proliferation.

Death, as resulting from hyperæmia solely, is rare; it is conceivable that it might follow an extension of the affection to the important centres in the medulla oblongata and cervical medulla. But, as a rule, the danger to life depends on hemorrhage, softening, and other changes of the spinal cord.

Diagnosis.

A demonstration of the fact that hyperæmia can exist in the spinal cord and can be recognized by its symptoms is not required. Although its existence is often very hard to demonstrate in the dead body, or quite impossible, yet is its existence in the highest degree probable, besides being proved by a number of clinical observations which are hardly susceptible of any other interpretation.

Not all, nor nearly all, the cases known in literature by this name are to be considered as deserving it; few of the uncomplicated cases which end in death can be reckoned as hyperæmia; and if nothing is discovered at the autopsy of such patients, the cause, doubtless, lies in defective methods of observation. It certainly seems to us improper to interpret as simple hyperæmia the large class of cases with severe and threatening symptoms (*e. g.*, the case of Desnos, numerous cases in Ollivier, etc.); and the fact that nothing besides hyperæmia has been discovered in many instances of so-called acute ascending paralysis, after careful microscopical examination, is far from proving that the fatal result was due to hyperæmia.

The diagnosis of hyperæmia of the structures within the spinal canal is chiefly based upon the *slightness of the sensory and motor disturbances*, which very rarely become severe; upon the *frequent and rapid changes in symptoms*, especially as regards their location; upon their *non-febrile, usually short and favorable course*, and upon the success of treatment suited to relieve hyperæmia.

It is obvious how uncertain these signs are, and how hard it is to separate them from those of slight inflammation or functional weakness. It is the duty of the future to throw light upon the matter. For the present, the following hints must suffice for the differential diagnosis. Spinal hyperæmia is distinguished from

Concussion of the spinal cord, by its comparative slowness of access, mildness, and rapid disappearance;

Spinal meningitis, by the absence of spasm of the back and neck, fever, and pains on moving the limbs;

Acute myelitis, by the absence of fever, severe symptoms of paralysis, contractures, paralysis of the bladder, and bed-sores;

Spinal apoplexy, by the comparatively slow development, slightness of the paralytic symptoms, and rapid termination in recovery;

Spinal anæmia, chiefly by the fact that lying on the back gives relief to the symptoms of the latter.

Thus, by exclusion and by observation of the course of the disease, we shall in many cases attain a tolerably certain diagnosis.

The *prognosis* of spinal hyperæmia must be regarded as favorable upon the whole. Serious complications may, of course, render it gloomy. In habitual, frequent hyperæmia, if the cause continues to exist, if the walls of the blood-vessels possess little power of resistance (involving the danger of hemorrhage), the prognosis will, of course, be serious.

Treatment.

Everything must first be tried, to fulfil the *causal indication* –in regard to which, detailed directions will not be required. The most favorable cases are those where we can ascertain that a discharge has been interfered with, or venous congestion exists, or exposure to cold has occurred; the remedies for these are obvious.

As a direct measure against the hyperæmia, the patient should be made to take a *suitable position*, avoiding the back, and preferring the side or face, with the extremities as low as possible. Next in order, *blood-letting* is the remedy most recommended. Venesection will be practised only when the symptoms are very violent—in plethoric, robust persons; a much better plan is to draw blood from the region of the spinal column, or from the anus, vagina, cervix uteri, etc., according to the nature of the case. From ten to twelve moist cups along the vertebral column, or a corresponding number of leeches, repeated according to circumstances at fixed intervals, will be most useful.

The attempt has also been made to treat spinal hyperæmia by *derivation to the skin*. A great variety of remedies may be used; the physician may select according to his patient's special case, but the indications are, unfortunately, very unsettled. The cold-water treatment has certainly a considerable sphere. For active hyperæmias, the hydro-therapeutists recommend milder procedures, as cool affusion and wraps to the back, soothing frictions, and sitz-baths; for the passive form, a more energetic course, including cold affusions and douches, exciting sitz-baths, energetic cold packing, and sea-baths; for such cases the gaseous thermal brine-baths (Rehme and Nauheim) seem especially suited.

A direct derivation of the blood to the skin is obtained by hot foot-baths with mustard and the like, by the warm douche applied several times a day to the back, by washing with a cold and a hot sponge alternately, by moderately warm full baths.

Sinapisms, blisters, etc., may also be of use in some cases.

A direct action upon the vessels of the cord has been attempted by applying Chapman's *ice-bags* to the back, and by the *galvanic current.* The indications for these measures are not precise, and it will be necessary in each case to be governed by the success that seems to be obtained.

Derivation to the intestine is also much favored in the form of saline purgatives, or, in more chronic cases, of the saline medicinal springs (Homburg, Kissingen, Marienbad, Karlsbad, etc.).

Among *internal remedies*, ergotin and belladonna should be tried by preference; the former must be given in large doses (Hammond).

Diet and regimen must be governed by personal circumstances; everything which might increase the hyperæmia (especially coitus) must be avoided.

2. *Hemorrhages of the Membranes of the Cord—Hæmatorrhachis—Meningeal Apoplexy.*

Ollivier, l. c. 3e éd. I. p. 465; II. pp. 90–137.—*Hasse*, l. c. 2. Aufl. p. 664.—*Hammond*, l. c. 3d edition. p. 440.—*Leyden*, Klinik der Rückenmarkskrankheiten. I. p. 367.—*M. Rosenthal*, l. c. 2. Aufl. p. 274.

Fallot, Hémorrhag. méningée spinale sousarachn. Arch. gén. 1830. T. XXIV. p. 438.—*Boscredon*, De l'apoplexie méningée spinale. Thèse. Paris, 1855.—*Ch. Bernard*, Observ. d'hémorrh. rhachid. Union méd. 1856. No. 62.—*Jaccoud*, Les paraplégies et l'ataxie. Paris, 1864. p. 232.—*Levier*, Beiträge zur Pathologie der Rückenmarksapoplexie. Diss. Bern. 1864. (Contains a copious list of authorities).—*Rob. Jackson*, Case of Spinal Apoplexy. Lancet. July 3. 1869. —*Hayem*, Des hémorrhag. intrarhachidiennes. Thèse. Paris, 1872.—*Rabow*, Fall von Meningealapoplexie in Folge von übermässiger Körperanstrengung. Berliner klin. Wochens. 1874. No. 52.

Definition.—Hæmatorrhachis implies *any effusion of blood n, about, or between the spinal meninges.* It occurs rarely, but n quite a characteristic manner. The vertebral canal does not resent favorable conditions for the production of hemorrhage, s has been reasonably explained by Hasse.

Etiology and Pathogenesis.

Little is known regarding the individual *predisposition* to eningeal hemorrhage. Most of the cases are observed in men. f diseases of the vessels of the meninges (fatty degeneration, theroma), we are mainly ignorant. The relation of hypertrophy f the heart to these hemorrhages has not been examined.

Among the *occasional causes*, the first to be mentioned are *urgical injuries* of the vertebral column, with or without direct esion of the membranes. Such hemorrhages have been observed o occur in consequence of stabs with swords and knives, fracures, contusions, and shocks of the column, a fall on the feet nd seat, or upon the arms and back of the neck, in new-born hildren after severe labor, etc.

Inflammatory and carious processes in the vertebræ have in ome cases led to lesion of the membranes and to bleeding from hem. *Congestions of the vertebral* canal and its contents (the auses of which have been enumerated in the previous section), specially those due to suppression of the menses or of a hemorhoidal discharge, are considered especially important causes of neningeal apoplexy. *Mental emotions* may act in a like manner y increasing the action of the heart.

Excessive bodily exertions are a frequent cause of meningeal leedings, probably through disturbances in the circulation: uch are the lifting of a heavy weight (Rabow), sudden violent novement, etc. Among these should be included the meningeal apoplexies which so often occur during the *violent spasms* of epilepsy, eclampsia, tetanus, trismus neonatorum, etc.

The *bursting of blood-vessels* or *aneurisms* into the vertebral canal has been repeatedly observed (Laënnec, A. Cooper, Pfeufer, Traube, and others).

Blood effused in the brain or *cerebral membranes* often passes down and fills the cavity of the spinal canal.

Of the occurrence of these bleedings in the various *hemorrhagic and infectious diseases* (scurvy, morbus maculosus, hemorrhagic small-pox, typhoid, etc.) little is known, except from a few reports of autopsies (see Hayem).

Pathological Anatomy.

It is necessary here to distinguish the various forms according to the position, distribution, and extent of the bleeding. We pass over those small ecchymoses and suggillations which so often accompany hyperæmia and inflammation within the vertebral canal.

Bleeding between the dura mater and the vertebral canal (in the extra-meningeal cellular tissue) is probably the most frequent cause. A dark, usually coagulated extravasation covers the outer surface of the dura to a varying depth, and infiltrates the cellular tissue between it and the vertebral canal, especially at its posterior wall. This extravasation may enclose the entire dura, but more usually it embraces it only partially; not rarely there are several separate foci. The dura is suffused with blood to a greater or less extent, and often covered with ecchymoses. The extravasation must be very considerable in order to produce a visible compression or change in the cord. It often extends around the nerve-trunks passing out of the vertebral canal.

Bleeding between the dura and the arachnoid (in the so-called arachnoid sac) is usually diffuse, very movable, partly fluid and partly coagulated, and usually consists of blood effused in the brain, though it may proceed from the bursting of vessels in the spinal meninges.

Of the hemorrhage accompanying many forms of internal pachymeningitis, producing hæmatoma of the dura mater, and situated likewise between the dura and the arachnoid, we will speak under Inflammations of the Dura Mater.

Bleeding in the arachnoid and pia (so-called subarachnoidal bleeding) is rare. It usually occurs in the subarachnoid cellular tissue, as the dense tissue of the pia seems little suited to extravasations. A more or less thick layer of dark-red, coagulated blood, infiltrated into the tissue of the soft membranes, surrounds the cord like a sheath, partly or completely, but in most cases it is limited in longitudinal extent, being confined to the length of one or two vertebræ. It may be found at various levels, and in all cases it inflicts severe injury upon the cord.

In all these forms of bleeding, the meninges usually exhibit but slight signs of reactive inflammation.

The cord itself may be more or less compressed, stained with red, softened, and (in the neighborhood of the seat of hemorrhage) hyperæmic. The same is true of the nerve-roots. The spinal fluid is turbid and of the color of blood.

Little is known regarding the other changes associated with extravasations. But it is hardly doubtful that in the spinal canal the color soon disappears, and the extravasation is partly reabsorbed and organized. Adhesion of the membranes, proliferation of connective tissue, and strong pigmentation of the membranes, are considered as the final changes.

Symptoms.

The disease usually *begins suddenly and violently*—often in the manner of apoplexy. The patient suddenly falls, with violent pains, almost always without serious disturbances of consciousness and sense. This mode of attack may be more or less complicated by the causes, by the nature of the injury, etc.

A *slow development* of the symptoms is rarer; with warnings of various kinds, symptoms of spinal congestion, lumbar pain, headache, for a longer or shorter time before the attack. Paresis then slowly appears, often unaccompanied by pain. The severity of the symptoms may increase after a few hours or days.

The characteristic symptoms are first those of *excitement*, caused by physical irritation of the meninges with their abundant nerves, the nerve-roots, and the cord itself, next those of *paralysis*, dependent on the pressure of the extravasation upon the cord and nerve-roots. According to the seat of the extravasation, it may press more upon the sensory or the motor portions.

The phenomena of *excitement* are the most prominent at the first, and chiefly consist of a violent pain, localized at a spot answering to the seat of the bleeding, and radiating in various directions, usually corresponding to the distribution of the nerve-roots first attacked. With this are conjoined *excentric sensations*, pain, formication, burning, tingling, etc., in the same regions ; hyperæsthesia is also occasionally mentioned, but this seems rather to belong to the period of reactive inflammation.

Symptoms of *motor excitement* appear at the same time, which seem to be of characteristic importance. Spasmodic jerking of the muscles, occasionally increasing to complete convulsions, trembling of the extremities, tonic tension and contracture of various groups of muscles, are the chief of these. They may become so active and so prominent that a special form of "convulsive" meningeal apoplexy has been distinguished. These spasms are partly deducible from direct irritation of the motor roots, and partly from reflex excitement.

The *vertebral column is stiff and painful* at this stage, making it very hard, or quite impossible, to rise in bed, sit, or bend. Great excitement and loss of sleep are caused by these pains and the muscular contractions.

The remarkable observation by Jackson proves that these symptoms of irritation are not necessarily always present ; in the case given by him they were entirely wanting.

It is not long, especially if the quantity of blood effused is considerable, before *paralytic symptoms* appear in the lower half of the body, but seldom reaching a great development, or amounting to complete paraplegia. As a rule, the patient has a furry feeling, numbness, sensations of swelling and heaviness in the limbs and trunk, with more or less distinct anæsthesia in the same parts. A feeling of very great muscular weakness and exhaustion indicates the same disturbances in the motor sphere ; a more or less severe paresis is more frequent, and complete paralysis is rare. The distribution depends on the seat of extravasation.

Reflex excitability has been found depressed in a few cases, but this probably occurs only in the regions supplied by the nerve-roots directly affected, though it might be exaggerated in the regions posterior to such roots.

Weakness of the bladder and rectum is rarely mentioned, but is usually present in severe cases.

Fever is absent, at least at the beginning of the disease; it may appear during the irritative reaction which sets in on the second or third day, but never becomes excessive.

According to the various levels occupied by the extravasation in the cord, the symptoms differ a little. The following symptoms indicate that the *cervical* region is affected:

The attack begins with pain in the arms and shoulders, with stiff neck and pain in the occiput; anæsthesia and paralysis most marked in the upper extremities; oculo-pupillary symptoms; difficulty of breathing and swallowing; violent dyspnœa; retarded and weak pulse.

The *dorsal region* is indicated when there is pain in the back and abdomen and pain in the form of a girdle, stiffness of the dorsal part of the spine, paralysis of the legs and the abdominal muscles; retained reflex power in the legs.

The *lumbar region* is indicated when there is pain in the loins, tearing pains in the lower extremities, the loins, perineum, bladder, and genitals; stiffness of the loins; well-marked paralysis of the lower extremities, with loss of reflex activity; paralysis of bladder and rectum.

Course and Termination.—After a sudden or gradual beginning, the symptoms usually remain stationary for a certain time, and sooner or later take a turn for the better. The symptoms of reactive inflammation are seldom prominent, or they disappear among the other symptoms. Slight fever appearing on the second or third day, with renewed pains, more distinct hyperæsthesia, etc., should be referred to the same; after two or three weeks these symptoms usually disappear.

In the majority of cases the disease runs a favorable course, unless the nature of the cause or the complications prevent it. A gradual improvement in the symptoms occurs, the paralysis disappears, but partial anæsthesia and paralysis often remain for a long time. The whole course is usually run in a limited time; in a few weeks or months a tolerably satisfactory cure may be completed.

Death is not rare—often occurring in a few hours or days; as when the extravasation is at a high level, and produces disturbance of the heart and respiration, or when blood effused extends upward to the brain, or when the central nervous system suffers a sudden severe impression (shock). If the extravasations are considerable, the severity of the compression may lead to complete paraplegia, cystitis, bed-sores, etc., and death may occur at a late stage.

Diagnosis.

The diagnosis of a meningeal hemorrhage is not always possible. If other severe diseases of the nervous system are present (hemorrhage of the brain, tetanus, convulsions, injury of the cord), it will not be in our power to recognize the complicating element of meningeal apoplexy, except under very peculiar circumstances. In such cases it will usually fail to be recognized, but that is seldom of much consequence.

The diagnosis of idiopathic and uncomplicated meningeal apoplexy can, however, be established in many cases.

It is chiefly based upon the *sudden occurrence of the symptoms*, upon the *peculiar combination of symptoms of meningeal irritation and spinal paralysis*, upon the absence of severe cerebral symptoms, the paraplegic character of the symptoms at the outset, the speedy improvement in the more severe symptoms, and the usually favorable termination. If we know the cause, that knowledge will often assist the diagnosis.

There is a series of spinal troubles, which closely resemble meningeal apoplexy in their symptoms, and are hard to distinguish from it; such are:

Commotio medullæ spinalis (shock), in which the symptoms of spasm are wanting, and the paralysis reaches its highest point at the onset. It should be remembered that commotio and meningeal apoplexy may occur together.

Bleeding in the substance of the cord (spinal apoplexy) is usually followed by severe palsy, including especially a high degree of anæsthesia; the pain and tendency to spasm are less marked; the latter is said by Brown-Séquard to be always absent in hæmatomyelia. This lesion usually causes rapid death, or incurable paralysis.

Meningitis and *myelitis* can usually be distinguished with readiness; they do not develop so rapidly, or if they do, they are always accompanied by fever. But the central myelitis which begins with fulminant symptoms (see below, under Myelitis) may give rise to confusion. In the latter, however, severe anæsthesia is never wanting, and the paralysis, also, is usually complete from the beginning.

For the diagnosis of the *seat* of the hemorrhage, the points given above may suffice; it may be ascertained from the distribution of the symptoms of palsy or irritation.

The *prognosis* is always dubious; but if the causal injury be not especially severe, and the extent of the hemorrhage not very great, it may be considered as comparatively favorable. We may say that if the first few days are passed in safety, the prognosis will become more favorable.

It is an unfavorable circumstance when the bleeding is very considerable, or is seated in the cervical region, or when marked reactive symptoms occur, or severe paraplegic symptoms, cystitis, bed-sores, etc.

If the bleeding is of small extent and the symptoms correspondingly mild, if the reaction is moderate and the patient young, all these circumstances are in the patient's favor.

Confinement to the bed of one or two months' duration, followed by a convalescence of several months, may be predicted.

Treatment.

Much can be done by way of prevention; by treating hyperæmia of the cord, or spasms, by regulating the menses, the hemorrhoidal bleeding, etc.

When the symptoms of meningeal bleeding have occurred, the first thing to be prescribed is *absolute rest in a proper position* (upon the side or face). Then the bleeding must be prevented from extending; the usual remedies consist of the *energetic application of ice to the vertebral column*, repeated *powerful purges*, and full *local bloodletting* (on the spine or anus). The effect of these may be aided by hot applications to the extremities, and by the internal or subcutaneous exhibition of large doses of ergotin. The diet must of course be properly regulated. Venesection can only be justified by very special circumstances (great plethora, violent action of the heart).

If symptoms of inflammatory reaction appear, the local bloodletting along the spine may be repeated, and as Leyden recommends, small portions of mercurial ointment may be rubbed in, and calomel given in subdivided doses.

The period of resorption demands special attention; it may be hastened by the internal and external use of iodine, by lukewarm baths, by a proper cold-water treatment, by the galvanic current. In later stages, the patient's strength may be supported and restored by tonics (quinia) and nux vomica.

In many cases a symptomatic treatment is demanded; thus, in the beginning, for the relief of pain and spasm (narcotics, etc.); later, for anæsthesia and paralysis (electricity); for cystitis, bedsores, etc

3. *Inflammation of the Spinal Dura Mater—Pachymeningitis Spinalis—Perimeningitis.*

Ollivier, l. c. II. pp. 272, 280. 3d ed.—*Hasse*, l. c. 2. Aufl. p. 689.—*Leyden*, l. c. pp. 385–406.—*M. Rosenthal*, l. c. 2. Aufl. p. 279.

H. Koehler, Monographie der Meningitis spin. 1861.—*Ruehle*, Klin. Mittheilungen. I. Bd. Zur Compress. des R.-M. Greifsw. med. Beitr. I. p. 5. 1863.—*Traube*, Deutsche Klinik. 1863. No. 20; Gesamm. Abhandl. II.—*Mannkopf*, Berlin. klin. Wochenschr. 1864. Nos. 4–7.—*A. Meyer*, De pachymeningitide cerebrospin. interna. Diss. Bonn. 1861.—*Th. Simon*, Ueber den Zustand des R.-M. in der Dementia paralytica. Archiv f. Psych. u. Nervenkrankh. II. 1869. p. 137, 143, 347.—*R. H. Mueller*, Ueber Peripachymeningitis. Diss. Königsberg. 1868.—*E. Wagner*, Arch. der Heilkunde. XI. 1870. p. 322.—*Charcot*, Pachyméningite cervicale hypertrophique. Soc. de Biol. 1871. p. 35; Gaz. méd. de

Paris, 1872. No. 9; Leçons, etc. 2ᵉ série. 3ᵉ fasc. p. 246. 1874.—*Joffroy*, De la pachymén. cervic. hypertroph. Paris, 1873.

The inflammations of the spinal dura mater, though known for a long time, have not been closely studied till quite lately. The significance of the disease has been better recognized, and a sharper line of separation from the other forms of spinal meningitis has been drawn, since it has been observed to occur isolated and spontaneously.

It is true that there exist but very few good observations; for this reason, the symptoms and diagnosis are very imperfectly made out. But we may properly make a distinction of two forms, according as the outer surface of the dura is preferred, and the morbid products are deposited between the dura and the vertebral column (external pachymeningitis), involving the loose cellular tissue, or as the inner surface is attacked, and becomes the seat of deposits (internal pachymeningitis).

a. Pachymeningitis Spinalis Externa—Peripachymeningitis.

Definition.—This disease consists of inflammation of the outer surface and layers of the dura and of the cellular tissue surrounding it. The morbid products, exudation, pus, connective tissue, are deposited between the dura and the vertebral column. This form of meningitis has been studied in but very few cases, and is in much need of further examination.

The chief cause of this form of inflammation consists in the presence of *inflammation of the vicinity*, which extends to the dura and the cellular tissue outside of the meninges. This is quite certainly the case in vertebral caries, and in *deeply ulcerated bed-sores*, which, especially when seated on the sacrum, easily provoke irritation of the structures within the cord. A similar thing, however—extension of the purulent and phlegmonous process of inflammation into the canal—has been observed in purulent inflammation of the dorsal muscles and the psoas (Traube), in inflammation in the connective tissue of the neck (Mannkopf), in the subpleural cellular tissue (H. Mueller), and all possible forms of chronic inflammation of the abdomen and thorax, especially peripleuritis, and also neuritis migrans, have been named as possible causes of peripachymeningitis.

It seems to us that this view of the secondary nature of the disease is carried quite too far. The observations which are quoted in proof certainly leave much room for doubt. Traube's cases are explained by him upon the supposition that the pachymeningitis externa was the primary complaint, the suppuration extending thence into the muscles of the back. This is quite in harmony with the clinical course and the results of autopsies. It is more natural and probable, *a priori*, that pus should force its way out through orifices in the narrow vertebral canal, with its rigid walls, and should afterwards spread in this direction or that, than that suppuration of several muscles should find its way simultaneously into the vertebral canal. There is a case in the *Medical Times* for January 6, 1855, p. 19, unfortunately reported very incompletely, which seems to confirm this view. An observation by Ollivier[1] seems to us of still more importance. In Mannkopf's case the secondary nature of the peripachymeningitic lesion could only be made extremely probable. R. H. Mueller's case cannot prove anything, as the connection between the peripleuritic membranes and those of the peripachymeningitis was in no way proved, and there were also found similar membranes, quite isolated, on the outer surface of the cerebral dura mater. The case reported by Leyden[2] lacks the confirmation of an autopsy.

We are therefore probably justified in claiming the possibility of the spontaneous and primary origin of pachymeningitis externa, especially as there is no solid objection to it.

We must await the result of further observations, for information as to whether surgical injuries, exposure to cold, syphilis and other causes may not originate this disease.

Pathological Anatomy.

More or less of the spinal dura mater is thickened; its outer layers are separated by inflammatory exudations, infiltration with cells, etc. This alteration is usually confined to short portions of the cord, the level of a few vertebræ, but it may extend over the greater part of the dura.

On the outer surface there is found a more or less abundant exudation of varying thickness; it has been found as much as half an inch thick (Ruehle). This consists either of pus, either fluid, or dry and caseous, enclosed in thickened connective tissue, and with the extrameningeal cellular tissue infiltrated; or of a soft, plastic, reddish gray young connective tissue, very vascular, sometimes covered with pus and full of small abscesses, sometimes undergoing cheesy degeneration. The last is the usual case in pachymeningitis resulting from vertebral caries (Michaud); there is a fungoid growth, originating from the outer surface of the dura, which is stimulated to produce the growth by the presence of carious pus.

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[1] II. p. 260. 3d ed.

[2] Loc. cit. p. 391.

The essential disease is, therefore, an inflammation of the outer layers of the dura and the surrounding cellular tissue, with exudation of a purulent, plastic, tuberculous, or other material.

The inner surface of the dura is also often thickened and opaque, often covered with a delicate fibrinous deposit. The pia and arachnoid seldom participate, but they have been seen adherent to the dura, opaque, and infiltrated with pus.

The cord itself is more or less compressed, flattened, pale, anæmic; often softened, full of microscopic fat-granules and granular corpuscles, and presenting, more frequently than is supposed, the signs of transverse myelitis. In the neighborhood of the place compressed, red softening and hyperæmia are found; in more chronic cases, ascending and descending secondary degeneration of the white columns (upwards in the posterior columns, downwards in the lateral).

The nerve-roots which pass out at the seat of pachymeningitis are compressed, atrophied, inflamed, and soft.

Add to these the anatomy of those processes which have acted as causes of pachymeningitis, or have accompanied it (vertebral caries, peripleuritis, muscular abscesses, phlegmons, etc.).

Symptoms.

The usual character of the disease is like that which will presently be described under Leptomeningitis, a more common affection. We therefore shall give but a short account of the chief symptoms in this place. The most important are as follows:

Pain in the back, various in seat and extent, according to the location of the disease. *Stiffness of the vertebral column*, which renders it difficult and painful to sit up. *Tension and jerking in various groups of muscles. Excentric pains*, in the form of a girdle or shooting into the extremities; sensation of a cord tied around; formication and *slight hyperæsthesia of the skin.*

To these are added, sooner or later, the symptoms of compression of the cord, gradually increasing; paralysis of various degrees, sometimes more marked in the motor, sometimes in the sensory sphere, sometimes in both at once; muscular tension, increased reflex action, especially those connected with tendons; paralysis of the sphincters, and bed-sores. These symptoms are due, partly to compression, partly to the myelitis which complicates it.

According to the nature of the original disease and the complications and secondary changes, disturbances of the general health, fever, and many derangements of internal organs may occur.

The symptoms of pachymeningitis may develop in an acute or a chronic way; in the acute (purulent) forms the symptoms of irritation are the more prominent, while in the chronic (plastic) forms these recede into the background, and the symptoms of compression of the cord dominate.

Of the *course* of pachymeningitis externa nothing can be said with definiteness, in the present state of our knowledge. The unfavorable cases which come to an autopsy cannot indicate the average event, for it is impossible to estimate how many have run a favorable course without being recognized in our present uncertainty regarding the diagnosis. We certainly know that the form in which it so frequently accompanies caries of the vertebræ is quite frequently arrested and partially recovered from, since the paralytic symptoms caused by it disappear. We have learnt only thus much: that in the severer cases the course may be various, but usually is protracted, and does not terminate unfavorably nor lead to a slow convalescence, until after the lapse of a considerable number of weeks.

The *diagnosis* is founded chiefly on the causal agencies we may be able to discover, and the associated, slowly increasing symptoms of meningeal irritation and of compression of the cord. The most difficult thing will always be to distinguish it from the other forms of meningitis. There is a diagnostic point —though of very doubtful value—which consists in the fact that external pachymeningitis but rarely ascends as high as the upper cervical region, and is therefore seldom accompanied by stiffness of the neck. In most cases, however, it will be impossible to decide whether the dura alone is affected, or whether the other meninges of the cord are also inflamed.

The *prognosis* follows from what has been said regarding the course of the disease. In forming it we shall be obliged to pay especial attention to the causal element.

In the *treatment* we ought principally to aim at a removal of the original lesion; if we succeed in curing this, we greatly improve the chances of curing the pachymeningitis. The special directions for this purpose need not be given here.

For the relief of pachymeningitis we proceed as is directed in the section on Leptomeningitis. It is of special importance to procure such relief in vertebral disease, where the improvement of the paraplegia depends upon the removal of the meningeal affection. In addition to treatment directed against the vertebral disease, the measures regarded with most confidence are the energetic use of brine-baths (bathing and drinking), the use of iodide of potassium and iodide of iron, pencilling the back with iodine, frictions with mercurial ointment, and the like. The white-hot iron, a very old remedy, has recently been warmly recommended for obstinate cases by Charcot.

b. Pachymeningitis Interna (Hypertrophica et Hæmorrhagica).

Definition.—Inflammation, chiefly of the inner surface of the dura; deposition of morbid products (exudations, extravasations, proliferation of connective tissue) on its inner surface, between dura and arachnoid. Frequent implication of the arachnoid and pia.

Two principal forms of this disease are known, possessing to a certain extent clinical characteristics; a simply hypertrophic form, leading to thickening of the dura (and usually of the finer membranes also) with connective tissue; and a pseudo-membranous, hemorrhagic form, characterized by a more or less abundant extravasation of blood. But few observations have been made as yet upon either.

Etiology.

The causes usually assigned are exposure to cold, and dampness of the dwelling (Charcot, Joffroy). Excessive use of alcohol seems to have some effect.

As to the *hemorrhagic form*, it seems to be established that it usually accompanies the affection of the same name in the cerebral dura mater, hæmatoma duræ matris, and therefore owns the same causes. The disease has not infrequently been found with psychical disorders, especially dementia paralytica (Simon, A. Meyer); also as a consequence of continued abuse of alcohol, of which Magnus Huss, Magnan, and Bouchereau have cases. Finally, Leyden describes a traumatic form; but the case adduced to illustrate it is not above doubt, as the patient was a drinker, and had suffered from symptoms of disease before the fall which caused the fracture of his skull; it is, therefore, not quite certain whether the fissure was the cause of the internal hemorrhagic pachymeningitis. A. Meyer says that both his cases occurred in cavalry soldiers, a class which is exposed to frequent falls and shocks.

Pathological Anatomy.

In the hypertrophic form we find a great thickening of the dura, chiefly caused by a considerable proliferation of its inner layers, which change into a hard cicatricial mass of connective tissue, with usually a distinct concentric arrangement of layers. Usually there is an intimate adhesion to the soft membranes, which are also thickened and proliferated, and form one continuous mass with the thickened dura mater; often, however, they are comparatively unchanged.

Thus there is formed a more or less extensive deposition, which compresses the spinal cord from one side or the other (usually from the rear), or which embraces it like a ring for a greater or less distance. The cord is sometimes simply compressed, pale, and soft, but more usually it presents all the marks of transverse myelitis of various extent, with secondary degeneration, formation of cavities, etc. The nerve-roots which are involved in the disease are wrapt up, compressed, and often in a state of advanced atrophy. The muscles which belong to them possess the microscopic characteristics of the usual degenerative atrophy.

In the hemorrhagic form the dura is covered to a greater or less extent with a mass of exudation, soft, rusty-brown in color, composed of fibrin and connective tissue, which is strewn with numerous extravasations of blood, and not seldom contains one or several large sacculated masses of blood. These masses contain dirty-brown, decomposed blood, numerous blood-crystals, pigment, detritus, etc. The enveloping mass of exudation is, in many places, yellowish, easily broken or torn, is disposed in layers, is attached but slightly to the dura or the arachnoid, and possesses an abundance of vessels.

These sacs of blood may vary in size and number. The hemorrhagic false membrane often extends over a great part of the cord, entirely enveloping it.

The process is exactly the same as that of hæmatoma of the cerebral dura mater; it is probable that the fibrinous inflammation is the primary element, and the bleeding only secondary. Yet it is not impossible that a primary bleeding may give rise to a subsequent inflammation.

The condition of the cord is as in the other form. The pia is usually tinged with blood, and so is the abundant spinal fluid.

Symptoms.

The *hypertrophic* form is at present thoroughly understood only as occurring in the cervical region, which seems to be its favorite seat; when situated there it has a somewhat characteristic course, and is described by Charcot as "pachyméningite cervicale hypertrophique."

He makes a first stage of the *symptoms of irritation*, which lasts two or three months, and is chiefly characterized by *pains*. These are very violent in the back of the neck and head, the shoulders, and arms ; are continuous, and from time to time experience exacerbations ; are often connected with a painful sensation of being bound with a cord in the upper part of the chest. *The neck is stiff*, but the spinous processes are not specially sensitive to pressure. *Formication* and *numbness*, and sometimes also *slight weakness of the upper extremities*, appear at this stage. *Trophic disturbances of the skin* are not infrequent—eruptions of herpes, development of vesicles on the upper extremities. Nausea and vomiting are but rarely observed.

The transition to the second stage, chiefly characterized by *paralysis* and *atrophy*, is very gradual. The upper extremities are more or less completely paralyzed, especially the district of the median and ulnar nerves, while in all cases hitherto observed the radial region has remained comparatively free. The result of this is the formation of a peculiar *position of the hand in extension, the fingers being held like claws.* This paralysis is conjoined with severe and quite uniform *atrophy*, so that the general aspect of the case reminds one of progressive muscular atrophy. The faradic excitability of the muscles is diminished or suspended. *Contractures of the muscles* appear, and single *spots of anæsthesia* of greater or less extent.

All this is doubtless mainly the result of the affection of the nerve-roots.

At a later period, *paralysis and contracture of the lower extremities* occur. Atrophy, however, is not associated with the affection in these parts, or not until a very late period. In severe cases complete *paraplegia* with marked anæsthesia, *paralysis of the bladder*, *bed-sores*, etc., may arise, and bring on the fatal termination.

These severe symptoms are doubtless to be referred to transverse myelitis at the spot compressed, and descending degeneration of the lateral columns, originating at that point. But the course is not always so bad ; there are cases in which the symptoms cease to make progress, or even are distinctly improved; but the disease is always chronic.

The symptoms of *hemorrhagic* pachymeningitis interna are still very obscure, and in most cases are complicated by those of the coexistent cerebral affection. The usual symptoms of a *slow* *neningitis, with periods of exacerbation*, pain in the loins and ack, tearing pains in the extremities, stiffness of the vertebræ nd of the nucha, increasing weakness of muscles, sometimes rising to complete palsy and paraplegia, moderate contractures, yperæsthesia and anæsthesia of the skin in various degrees, veakness of the bladder, etc., are sufficient, in case *certain auses are present* (as drunkenness, paralysis, hæmatoma of the erebral dura mater), to arouse a suspicion of disease of the spial dura, and, at least in many cases, to lead to a probable diagosis.

We have yet to learn whether this disease, like the cerebral orm, is marked by aggravation of the symptoms from time to ime, and whether this fact can be made useful in diagnosis.

The disease, in this form, will usually take an unfavorable ourse.

The diagnosis may be inferred from the previous brief sketch f the two forms. Pachyméningite cervicale hypertrophique has certain resemblance to progressive muscular atrophy, atrophic ateral sclerosis (Charcot), etc. The most important points of istinction are the stage of pains, the partial anæsthesias, paralegia without atrophy, etc. It will not be always possible to istinguish it from meningeal tumors.

The hemorrhagic form is in need of a more exact diagnostic oundary.

The *prognosis* requires no remarks.

The *treatment* will resemble that of meningitis in general (see ext section). In more acute cases antiphlogistics of all sorts vill be of use.

In later stages and chronic forms we may have recourse to erivatives, preparations of iodine, galvanism, and the use of aths or the cold-water treatment. Prominent symptoms, as ain, paralysis, atrophy, etc., require a special symptomatic reatment.

4. *Inflammations of the Pia Mater and Spinal Arachnoid—Leptomeningitis Spinalis—Perimyelitis and Arachnitis.*

P. Frank, l. c. 1792.—*Ollivier*, l. c. 3d ed. II. p. 232.—*Hasse*, l. c. 2. Aufl. p. 690.—*Hammond*, l. c. 3d ed. p. 444.—*Leyden*, l. c. pp. 406–448.—*M. Rosenthal*, l. c. 2. Aufl. p. 283.—*Koehler*, Monographie der Meningitis spinalis. Leipzig. 1861. (Contains a great deal of valuable matter and very full references.)

Klohss, Diss. de myelitide. Halis. 1820. Hufeland's Journ. XVI. 1823.—*Fuchs*, Die Rückenmarksentzündung. Bamberg. 1825.—*Henoch*, Schmidt's Jahrb. Bd. 28. 1846.—*Evans Reeves*, Diseases of the Spinal Cord and its Membranes. Monthly Journ. of Med. 1855. p. 506; Edinb. Med. Journ. 1855-56. pp. 129 and 302.—*Noetel*, De meningitide spinali. Diss. Berlin. 1861.—*Beaumetz*, Méning. spinale, suivie de roideur des extrém. infér. Gaz. des Hôp. 1861. No. 129.—*Brown-Séquard*, Lectures on the Principal Forms of Paralysis of the Lower Extremities. London. 1861. p. 66, etc.—*Camerer*, Ueber Meningitis spin. chron. und deren Differentialdiagnose. Würtemb. Correspondenzblatt XXXII. 1862.—*Jaccoud*, Leçons de clinique médicale. 1867. pp. 372-420.—*Vulpian*, Note sur un cas de méning. spinale et de sclérose corticale annulaire de la moëlle ép. Arch. de Physiol. II. p. 279. 1869.—*Liouville*, Étude anatomo-pathologique de la méningite cérébro-spin. tubercul. Arch. d. Physiol. III. p. 490. 1870.—*Stokes*, Chronic Inflammation of the Spinal Cord and its Membranes. Dubl. Journ. of Med. Science. Vol. LVI. p. 62. 1873.—*Bruberger*, Fall von Meningitis syphil., etc. Virch. Arch. 1874. Bd. 60.

Compare also *von Ziemssen*, Meningit. cerebrospin. epidemica, in Vol II. of this Cyclopædia, and the full references there given.

Inflammation of the soft membranes of the spinal cord is the most important and the most frequent of spinal meningeal affections; and this is what is intended when spinal meningitis without further designation is spoken of. All treatises by the earlier authors relate to this. But a great deal has been thrown under this title which must certainly be separated from meningitis, or which occurs simultaneously with it. It is reserved for future investigations to show more clearly than has been done the line of demarcation between meningitis and myelitis; for this purpose accurate histological examination of the cord by the latest methods in cases of meningitis is absolutely needed. We have, however, very little positive information upon this point.

It would seem hardly probable that any considerable inflammation of the pia mater could exist without involving the cord to some extent. The pia distributes the vessels to the entire cord; from it pass out the processes of connective tissue which compose the framework of the latter; so that any inflammatory irritation affecting the vascular district of the pia must be felt more or less in the vessels of the cord, and when once a morbid process is established in the connective tissue of the pia, it is hard to see why it should not spread to some extent in the cord.

A certain degree of independence on the part of the two vascular districts of the pia and the cord must be admitted, as each may be affected singly; this fact is certainly established in relation to the cord, and suggests to us that the nervous elements themselves possess some influence upon the disease, and that they may be primarily attacked, or, at least, may bear a prominent part in the production and localization of diseases of the connective substance.

It is in diseases of the pia mater that we shall be obliged to pay special attention to the possibility of an implication of the cord. This has been done far too seldom, in my opinion; pathological anatomy has devoted too little attention to the point. The most that has been done has been to demonstrate an implication of the nerve-roots that pass through the diseased membrane; or an affection of the cord in very severe cases. Few observations have been made systematically and with the aid of delicate methods. Mannkopf, in epidemic cerebro-spinal meningitis, has seen abundant cell-infiltration following the course of the vessels even into the cord. Fronmueller has seen the central canal full of pus-cells in the same disease; Liouville, in tubercular meningitis, has seen tubercles in the processes of the pia, in the fissures of the cord, and Vulpian has demonstrated in one case of inflammatory thickening of the pia an annular sclerosis of the cord, extending to some depth, especially in the posterior columns, the dependence of which upon meningitis is, however, at the least doubtful.

This deficiency has been made good by the investigations of Dr. F. Schultze,[1] relating to three cases of leptomeningitis spinalis which show a very considerable implication of the nerve-roots and the cord itself. The principal results of these investigations is the following; for some further details the reader is referred to the pathological anatomy of acute meningitis.

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[1] Berl. klin. Wochenschrift. 1876. No. 1. Dr. Schultze has permitted me to view his microscopical preparations bearing on this point, and it is my agreeable duty to acknowledge gratefully the various assistance which the free use of his numerous and excellent preparations has rendered to me.

The *nerve-roots* are in a state of marked inflammation (infiltration with cells, especially in the neighborhood of the vessels, the nerve-fibres swollen, granular, beginning to break down, the axis-cylinders swollen and granular); the bundles of root-fibres which enter the cord are more or less considerably swollen. In the *cord itself*, there is found a peripheral interstitial myelitis (infiltration of the neuroglia with cells and nuclei) penetrating to a greater or less depth, or actual parenchymatous myelitis in large and small foci (enormously swollen axis-cylinders, cloudiness and granular decay of the medullary sheath, axis-cylinders breaking down, etc.). In the gray substance there were found signs of œdematous swelling in the ganglion-cells, but no other distinct changes. Only the central canal appeared closely packed with round cells, and its vicinity also infiltrated to a considerable distance with the same elements.

It certainly follows from these facts that we shall have to take a somewhat different point of view from what has previously been customary in explaining the origin of the symptoms of spinal leptomeningitis. It is quite clear that the inflammation of the pia can produce no very marked symptoms by itself; the principal would be that of pain and the consequent reflex phenomena, owing to its abundant supply of nerves. But the most important and essential symptoms must arise from an affection of the nerve-roots and the cord itself; and for this reason it will be very desirable to distinguish between purely meningeal and purely spinal symptoms, as well as those referable to the roots. A delicate clinical analysis will be required to distinguish these groups of symptoms.

I have premised these remarks in order to facilitate the understanding of the symptoms of meningitis, and to indicate the defects in our knowledge.

As for distinguishing the inflammation of the pia, the so-called perimyelitis, from arachnitis, I consider it impossible, both upon pathologico-anatomical and practical grounds.

Finally, the best division that we can make in practice—in pite of the great variety of form which spinal leptomeningitis nay take—seems to me to be that which recognizes but two chief orms, the *acute* and the *chronic*.

a. Leptomeningitis Spinalis Acuta.

By this title we understand a *febrile inflammation of the soft nembranes* (pia and arachnoid) *of the cord, beginning with vioent symptoms* and characterized chiefly by an exudation of a urulent-fibrinous, more rarely a sero-fibrinous nature. It occurs ost frequently in an epidemic form, and in association with the orresponding affection of the cerebral membranes, as cerebropinal meningitis. But it also occurs sporadically; and this orm is that which will occupy most of our attention.

Etiology and Pathogenesis.

The *predisposition* to acute spinal meningitis is quite general, lthough the causes upon which it depends are not yet adeuately known. It attacks by preference children and young ersons, and the male sex; it is more frequent when there is tenency to scrofulosis and tuberculosis; all sorts of weakening nfluences (bad dwellings, poor food, sexual and other excesses, tc.) increase the tendency. As regards the way in which these auses act upon the spinal membranes, we know nothing precisely.

Among the *immediate causes*, exposure to cold certainly lays a very important part. It has often enough been observed hat sleeping on damp ground or snow, or the action of a cold ind striking upon the back while sweating, or an unexpected all into the water, and other similar occurrences, have led to an ttack of acute leptomeningitis. But in this case, as in most inlammations of internal organs caused by cold, we know nothing ith certainty of the delicate mechanism of the entire process.

The action of the sun's heat, *insolation*, upon the back is a ery questionable source of acute spinal meningitis.

Surgical lesions, however, constitute an unquestionable and ery frequent cause. The disease has been observed after simple concussion of the spine, as in falling down stairs; after cutting, stabbing, and gunshot wounds of the spinal column and its contents; after luxation and fracture of the vertebræ; after the operation for spina bifida, etc.

Inflammations and other affections of neighboring parts are often propagated to the spinal meninges and arouse inflammation in them; thus caries of the vertebræ, acute inflammation of the dura spinalis and the extra-meningeal cellular tissue, deep bed-sores reaching to the cavity of the sacrum; bursting of cavities in the lungs into the vertebral sac; carcinoma of the vertebræ; and finally, acute inflammations of the cord itself, which extend to the pia. The most frequent cause among this class is the acute *inflammation of the cerebral pia mater*, which is usually complicated with that of the spine. The spreading is usually effected through the open communication between the cerebral and the spinal cavities, through the anatomical continuity of the meninges, the current of the cerebro-spinal fluid, the gravitation of the inflammatory products, extravasations, etc., into the spinal cavity, and there setting up an inflammatory irritation; and finally, it is doubtless due to other causes acting upon both the cerebral and spinal meninges at once. Thus it is easily understood how the inflammation of the cerebral pia mater so often extends downwards to the spinal pia mater.

Of *tubercular basilar meningitis*, according to recent observations, a tubercular spinal meningitis is a quite regular accompaniment. Hence the appearance of spinal symptoms in this disease.

The publications of Weber[1] and Bierbaum[2] are of comparatively little force as evidence. The most important cases are three by Koehler,[3] which demonstrate the coexistence of tubercle in the pia mater of the brain and of the cord. Also two cases presented by the same author in his monograph.[4] Liouville has recently stated that the occurrence is a very regular one, and that among numerous cases, he has seen it in every one. In F. Schultze's three cases, above mentioned, the spinal pia participated each time in the cerebral inflammation. Leyden,[5] also, gives a case of this kind, without making any exact statements regarding the frequency of its occurrence. It is certainly much more frequent than has been supposed.

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[1] Deutsche Klinik. 1852. No. 34. p. 380.

[2] Journ. f. Kinderkrankh. Bd. 26. p. 355. 1856.

[3] Ibidem, Bd. 32. 1859. p. 409.

[4] Loc. cit. p. 127.

[5] Loc. cit. p. 438.

Among the somewhat doubtful causes of acute spinal meningitis may be mentioned *dentition, suppressed perspiration of the feet, suppression of the menses and of hemorrhoidal bleeding, disappearance of acute exanthemata*, etc., although instances of all these are given in the older literature.

The disease also occurs now and then in company with, or during convalescence from *febrile diseases* (pneumonia, acute articular rheumatism, etc.), or *infectious diseases* (acute exanthemata, cholera, typhoid, etc.). But this seems to apply chiefly to the epidemic variety. In *child-birth*, Koehler has repeatedly observed acute spinal meningitis.

Finally, we must speak of *epidemic and infectious influences*. The form of spinal meningitis which originates in company with cerebral meningitis under the action of some as yet unknown infectious material, and has repeatedly during the present century assumed the dimensions of a wide-spread epidemic, is by far the commonest and the most important. We can only refer to von Ziemssen's admirable delineation of the disease, in Vol. II. of this Cyclopædia.

Gauné[1] reports the occurrence, in a girl's boarding-school, of a light epidemic of favorable termination, not going beyond the symptoms of hyperæmia of the cord in some cases. The immediate cause was unknown.

Pathological Anatomy.

The morbid changes found in acute spinal meningitis differ according to the stage of the disease. In general, three periods may be distinguished, which of course pass imperceptibly into one another: 1. A stage of *hyperæmia and commencing exudation*. 2. A stage of *serous or purulent fibrinous exudation*, and 3. A stage of *resorption* or *termination* in some other way. The one most commonly observed is the second; the first much more rarely, especially in the fulminant attack of epidemic cerebrospinal meningitis.

In the first stage, the pia contains a great deal of blood, is colored from a rosy to a dark red tint, is full like velvet, and in places is dotted with blood, or with ecchymoses of various size, while the blood-vessels are very full. The tissue is succulent, swollen, imbibed with serum, and the cerebro-spinal fluid is slightly turbid. The arachnoid shares in these characters to a greater or less extent; the hyperæmia usually extends also to parts of the dura of various extent, and is also to be seen in the substance of the cord itself.

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[1] Arch. gén. 1858.

The second stage comes on by degrees; the watery contents of the tissues increase, the spinal fluid becomes more and more turbid, fibrinous flocks and plates are formed in the subarachnoid tissue, or adhere to the surface of the dura; the pia is more and more opaque, the subarachnoid tissues swell to a gelatinous mass which more and more conceals the former redness. The purulent character of the exudation becomes more and more distinct; the opacity increases, the tint alters to a whitish yellow, or green-yellow, and at last the whole pia and subarachnoid tissue are infiltrated uniformly with pus. The spinal fluid, growing more turbid, assumes a sero-purulent appearance and contains numerous flocks of fibrin, some floating and some covering the free surfaces of the meninges. In some cases there is to be seen, in addition to the exudation, a variable number of small miliary nodules, gray or whitish yellow, chiefly distributed in the course of the vessels, in the pia, and arachnoid, but not infrequently strewn over the surface of the dura in considerable numbers (tuberculous meningitis). In this form the exudation is usually of a rather gelatinous consistency, serous, yellowish in color, and rarely constitutes a strictly purulent infiltration.

The *distribution of the exudation* varies greatly. Sometimes it is spread over a larger, sometimes over a smaller space, but it usually covers the greater part or the entire length of the cord; the posterior surface is apt to have more of it than the anterior, owing doubtless to the patient's lying on his back. It is evident from what has been said, that the exudation frequently extends to the membranes of the brain, though in very various amount. In such case a direct connection between the masses in the brain and cord can always be shown to exist along the base of the brain, although the quantity deposited on the medulla oblongata is often remarkably small.

The *arachnoid* is almost regularly involved in the inflammatory exudation. It is opaque and thickened, infiltrated with serum or pus, and often abounds with gray miliary tubercles; in such cases the subarachnoid connective tissue is always infiltrated similarly, and in its swollen condition forms a layer of exudation, surrounding the cord like a sheath. The *dura spinalis* is less frequently mentioned as involved, yet it is often hyperæmic, often opaque to a considerable extent, and covered with thin, fibrino-purulent exudation. In a few cases peripachymeningitic hemorrhages have also been seen.

The *nerve-roots* are always more or less involved in the inflammatory changes; they are enveloped in thick masses of exudation, swollen, softened, their fibrillation is indistinct, their consistency diminished.

Of the condition of the *cord* itself in acute meningitis our information is quite scanty; it has been found pale and œdematous, at other times more hyperæmic, but usually softened; the softening may be somewhat uniform, or it may be disseminated and limited to certain spots; in a few cases a purulent infiltration of the cord, of variable extent, could be distinctly seen with the naked eye.

Microscopical examination discovers in the soft membranes of the cord all the signs of exudative inflammation—abundant cell-infiltration, especially along the vessels, great fulness of the capillaries, swelling and spreading of the bundles of connective tissue, etc. Such tubercular nodules as may be present show the characteristic histological marks, and are chiefly found along the vessels. F. Schultze found abundant infiltration with cells in the walls of the vessels which run in the anterior and posterior nerve-roots; the infiltration extended to the neuroglia. Some of the nerve-fibres of the roots appeared altered, their medullary sheath being cloudy and granular, the axis-cylinders decidedly swollen and in a state of granular degeneration. The bundles of roots which passed into the cord seemed, therefore, to be thickened at many points, and could be followed for a short distance into the cord, where they resumed their normal dimensions and appearance in the neighborhood of the gray substance.

In the cord, F. Schultze distinguished two sorts of affection. Either there was infiltration with cells and nuclei, affecting chiefly the neuroglia, without direct implication of the nerve-fibres, and limited to the peripheral layers of medulla (peripheral interstitial myelitis), or the nerve-fibres were principally involved in the inflammatory processes (parenchymatous myelitis), so that distinct myelitic foci of various extent and position could be recognized. Thus there were found in the transverse section (especially in the lateral columns) narrow wedge-shaped regions of inflammation, with the point directed towards the centre, of which the longitudinal extent varied. Single nerve-fibres in the condition of inflammatory swelling were found at all parts of the transverse section. In the vessels of the cord no essential changes were found; in the gray substance not much that was abnormal could be seen, except an apparently œdematous swelling of many of the ganglion-cells; the central canal, however, was regularly obliterated, plugged with roundish cells, and its vicinity also infiltrated with similar round cells.

The second stage is that in which death usually occurs; hence its anatomical changes are the best known. In cases not terminating in death a third stage must be admitted, in which a complete restoration to the normal state occurs with complete resorption—a process which can only be observed by fortunate accident—or various permanent changes occur, residua and consequences of the acute process of very various significance, and not rarely developing in a chronic way. The most frequently seen are opacity and thickening of the soft membranes; adhesions of these membranes to one another and to the dura are not rare; large accumulations of fluid in the arachnoid space (hydrorrhachis); more rarely chronic processes develop in the cord which continue to increase—sclerosis and atrophy of the cord, either affecting single columns, or distributed in the form of islands of disease, or diffused through the whole medulla. We have, therefore, after acute spinal meningitis, either the pernicious changes, progressive chronic meningitis or myelitis, or else those comparatively harmless processes (opacity, thickening, calcareous plates, etc.), which are so often found in the body without having given rise to special symptoms during life.

The anatomical changes of the other organs during acute spinal meningitis require no special description here. They differ, according to the course of the disease and the manner of death, but do not present anything especial. The most important of them have been sufficiently described in Vol. II. under Cerebro-Spinal Meningitis.

Symptoms.

Characteristic as are the features of acute spinal meningitis, it is yet rarely found pure and isolated; it is especially common o find the cerebral pia mater affected at the same time, which omplicates the picture and hinders the precise interpretation of he symptoms. Yet with some attention and experience the haracteristic features of spinal meningitis can be deduced from he symptoms as a whole.

General Description.—Acute spinal meningitis usually begins suddenly; often with fulminant symptoms. Distinct and marked precursory symptoms are observed in only a minority of he cases; general heaviness and depression, slight chill, and a ittle gastric disturbance, fleeting pains in the head and back, estlessness and sleeplessness, preceding for a time the outbreak of more serious symptoms.

The proper *commencement* of the disease is marked by a more or less severe *chill*, directly followed by violent febrile symp-oms; the temperature is raised, without any regularity of curve, he pulse is full, hard, rapid, very rarely retarded at the outset. Vomiting and severe cerebral symptoms are observed only in the cerebro-spinal form.

The painful symptoms very soon become the most prominent; he chief one consists of an intense, deep-seated, boring *pain in he back*, which is much complained of. According to the local-zation of the inflammation, the pain may occupy various places loins, back, or nape of the neck), and have a various extent, but s increased less by pressure on the spinous processes than by movement of the vertebral column and the extremities; it is usually very violent, but remitting; and from the place where it s seated, pains shoot out around the trunk in a ring, or extend o the extremities, piercing them in all directions. Thus all movements are rendered extremely painful.

The pain in the back is regularly and characteristically asso-iated with a severe *rigidity of the spine*, most marked at the height of the disease. The best-known variety is that which ttacks the nucha, and is so constant in epidemics; if the disease s situated lower down, the lumbar spine may be the chief seat of the painful stiffness, and when the disease is diffuse, the rigid-ty, produced by muscular tension and contracture, may extend o the entire column, and produce a very close resemblance to etanic spasm.

Quite analogous phenomena are observed in the muscles of the extremities; painful tension and stiffness, not rarely increased to the point of extreme contractures, producing rigid immobility of the limbs; in some places spasmodic twitchings occur, which give the patient extreme pain, and somewhat resemble tetanic spasms, but seldom amount to general convulsions. Such twitchings, in single muscles, are apt to be produced by attempts to use them.

The *skin* of the extremities and the trunk, as far as its sensitive nerves originate from roots which are involved in the inflammation, is in a state of marked *hyperæsthesia*, so that every touch, every movement of the patient, calls out expressions of pain, even in cases where the consciousness is quite impaired. *Hyperæsthesia of the muscles* seems to be equally demonstrable in many cases.

The *reflex activity* is usually increased at the beginning, but diminishes during the course of the disease.

With these sensory and motor symptoms, *disturbances of the evacuation of urine and fæces* are early allied; these functions are performed with difficulty, owing, as is usually supposed, to a spasmodic condition of the sphincters; artificial means are often required in order to regulate them.

Disturbances of the *thoracic viscera* do not occur unless the cervical part of the cord is involved in the inflammation. In this case *difficulty of breathing* appears, due to the rigidity and painfulness of the muscles of respiration; in the advanced stages, serious symptoms of dyspnœa occur, which may increase to complete asphyxia. Disturbances of the *action of the heart* (great retardation or acceleration) may be added.

Cerebral symptoms, as vertigo, violent headache, delirium, unconsciousness, coma, etc., are not rare; when they occur, it is usually in cases in which some degree of implication of the cerebral pia mater is present. They may appear at an early period, or later in the course of the disease, and when fully developed, often indicate the probability of a fatal event.

As the disease progresses, the symptoms of irritation may become less and less prominent, and give place to more distinct *symptoms of paralysis ;* but both classes of symptoms may be intermingled in various ways. Pareses and paralyses occur; the immobility of the limbs is no longer owing to muscular tension, but to motor weakness; symptoms of vesical paralysis occur; the cutaneous sensibility is lessened, and even high degrees of anæsthesia may appear; at last, paralysis of the respiratory apparatus may lead to very threatening symptoms.

When the disease has reached this height, everything may rapidly go from bad to worse, and death may soon occur; the immediate cause of death is usually a progressive paralysis of respiration and circulation, associated with deep coma, and not rarely with a great rise of temperature during the agony.

At other times the course is more protracted; the severity of the symptoms lessens, deceitful signs of temporary improvement appear, while the disease, as a whole, continues to make progress; severe paralysis, bed-sores, etc., appear, and death follows after long suffering. A secondary implication of the cord is always to be supposed in such cases.

Improvement may really occur; in slight cases this is often wonderfully speedy, with a short convalescence, while in severe cases it is more gradual, and has many fluctuations. The convalescence is then tedious, the forces recover themselves but slowly, the abnormal irritability disappears very gradually, and the patient needs care for a very long time. Incurable injuries are often left behind; paralysis and atrophy of single muscles and groups of muscles, or of whole extremities, contractures, anæsthesia, etc. Signs of degeneration and sclerosis of single columns of the cord may continue after the disease is over.

Interpretation of the symptoms.—Among the most constant and important is, without doubt, the *pain in the back*. It is usually very severe, deep-seated, boring, and tensive, of various extent, but hardly ever absent. It is a special characteristic, that it is very much increased by every movement of the trunk or limbs, so that the patient is often compelled to maintain absolute repose from this circumstance alone. The movements connected with evacuation of urine and fæces also naturally increase the pain in the back. Pressure on the vertebral column does not always increase it.

This dorsal pain doubtless owes its existence to inflammatory irritation of the nerves of the pia and dura, to inflammation of the posterior roots, and the consequent hyperæsthesia, but hardly to an inflammatory implication of the cord itself.

Pains in the extremities, of a tearing or boring character, and increased by every movement, are seldom wanting, and have doubtless the same origin. They correspond in their location and extent with the inflamed portion of the cord.

To similar processes of irritation in the motor apparatus may be referred the equally frequent and important symptoms of *muscular tension, contractures, spasms, stiffness of the spine or neck*, etc. The most characteristic of all is the stiffness of the spine and neck, with the head drawn backwards, the back stretched, often in the position of opisthotonus, and stiff and hard, especially when active or passive movements are made. In the extremities, the extensors are most affected, but often the flexors; the limbs in this condition are often of the hardness of stone, and immovable. This tension relaxes a little at times, is especially increased by attempts at moving, but is usually not much increased by reflex irritation.

The pathogenesis of these motor symptoms is not yet settled. It is commonly believed that they originate in the *reflex way*, owing to the abnormal irritation of the posterior roots, and might be regarded as reflex contractures. This may be true to some extent and in some cases. It is, however, quite certain that the muscular tension is *half voluntary* in character, or is increased by a voluntary act, which has for its object the prevention of movement when all movements are so painful. The chief weight, however, should be laid upon *direct irritation of the motor apparatus*.

Such a condition may depend in part on inflammatory irritation of the anterior roots, in part on irritation of the motor paths in the lateral columns by secondary points of myelitis (as F. Schultze has shown). If the latter were the correct solution, it would lead us to infer that the fibres for the trunk-muscles occupied some exposed situation (possibly in the external periphery of the lateral columns), in order to explain the great amount of rigidity in these muscles and in those of the nucha. The assumption of a *direct irritation* of the motor apparatus is confirmed in an interesting manner by two cases in which Leyden[1] saw the muscles of the back of the neck paralyzed in the latter part of the disease. And, in fine, the clonic muscular twitchings which occur spontaneously, or during efforts to move, may best be explained by direct irritation of the motor paths.

It is hard to say how the *hyperæsthesia*, which is almost always present in a high degree, is to be explained. It is most marked in the skin, but extends also to the deeper parts, the joints, muscles, etc. It may be so severe that the slightest touch or change of position calls forth the strongest expressions of pain; even unconscious patients may shrink and make motions of defence, when taken hold of. All voluntary movements of the hyperæsthetic parts cause severe pain. The hyperæsthesia is usually most marked in the lower extremities and the lower half of the trunk, while it is less marked and more rare in the upper limbs.

We are not yet in a position to assign any cause for this hyperæsthesia except the inflammatory irritation of the posterior roots, and perhaps also an implication of the white columns of the cord. It must be admitted that this explanation is very inadequate.

The *anæsthesia* and *paralysis* which appear at a later stage are more intelligible. They vary much in form and distribution; there may be a paralysis of single muscles or groups of muscles, with or without atrophy; paralysis of an entire lower extremity, or paraplegia; the upper extremities are reached, or are the exclusive seat of paralysis, in few cases. In complications with cerebral meningitis, paralysis of some cerebral nerves, disturbances of the senses, etc., are frequent. These paralyses often disappear quickly as the disease improves, while at other times they improve very slowly, or may be quite incurable; and from these circumstances inferences may be drawn as to the nature of the causal lesion.

We cannot be much mistaken if we ascribe the majority of these sensory and motor paralyses to the demonstrably *severe affection of the posterior and anterior* roots. It requires no explanation to show that the swelling and opacity of the nerve-fibres and axis-cylinders may very easily reach a point which is incompatible with the further performance of their functions; that plastic infiltration of the neurilemma may so compress the root-fibres as to render them unfit for conduction; and that the embedding of the delicate bundles of root-fibres in a considerable mass of fibrinous exudation will very easily destroy the function of the roots. It is, however, also possible that the *myelitic points in the white columns of the cord*, especially in the lateral columns, may in the course of the disease become dangerous to various paths of conduction, and produce paralysis. And finally, we may remember that *the presence of a large fluid exudation in the sac of the dura* may compress the cord and the nerve-roots to such an extent as to produce paralysis. This lesion will become probable only under special conditions, as when the paralysis is diffuse and not very severe.

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[1] Klinik, etc. I. p. 417.

The symptoms on the part of the vegetative organs are less constant, and often hard to interpret; their physiological connection with the cord is well known to be undetermined in many points.

In the *urinary apparatus*, a frequent disposition to pass water, in connection with ischuria or complete retention of urine, has often been observed; it has been usual to explain these symptoms as the consequence of a direct or reflex spasm of the sphincter of the bladder. At a later stage, well-marked weakness and paralysis of the bladder often occur, usually accompanied by paraplegia. For the explanation of this we require the same facts which explain motor paralysis; the mechanism of the various forms is that which has been stated on page 79 *et seq.*

The composition and quantity of the urine seem to depend on the severity of the fever; it is at first saturated, dark, scanty, clouded with urates, but later becomes abundant, light, and clear. In a good many cases (especially of the epidemic form), an unusual *excess in quantity* has been observed, which is usually ascribed to a direct nervous stimulation of the secretion, originating in the cord; and in like manner the rarer affection, mellituria.

In the *digestive apparatus* we observe the disturbances which sually accompany febrile complaints, and in addition, most equently constipation, which is referred by Koehler to spasm f the intestinal muscles, and consequent interference with peri-altic action, and to the spasmodic tension of the abdominal uscles. This explanation may be correct in the first stages, ut at a later point, the sluggishness and weakness which are o characteristic of intestinal movements in many spinal diseases are more probably the cause. The abdomen is usually sunken and tense; swelling and meteorism are rare, and so is diarrhœa. *Vomiting*, which often occurs, is probably always due to a localization of the process at the basis of the brain, and therefore is most frequent in cerebro-spinal meningitis.

The *respiratory apparatus* is remarkably affected in all severe attacks of spinal meningitis, especially when located in the cervical region. All the degrees of accelerated and difficult breathing occur, up to the extreme of dyspnœa, and even asphyxia. The explanation is to be sought in the following circumstances: irritation of the cervical roots, and the consequent spasmodic tension and immobility of the respiratory muscles, or, at later periods, paralysis of the same apparatus; irritation or paralysis of the respiratory paths in the lateral columns of the cervical medulla; and finally, the direct affection of the medulla oblongata and the centres of respiration which it contains. Near the fatal termination, the Cheyne-Stokes phenomenon of respiration has been repeatedly observed, which probably always indicates that the medulla oblongata has been reached by the inflammation.[1]

The disturbances of the apparatus of circulation have been but little studied, and owing to their complicated origin, they are hard to interpret. They depend firstly and chiefly upon the fever. Increased frequency of pulse is the rule; yet retardation often occurs, especially in cerebral complications; great irregularity in rapidity and rhythm is often observed; in fatal cases, towards the close, as the temperature rises, the pulse often becomes too fast to count. It may be left to the reader to form for himself a suitable explanation of the disturbances in such a case, by reference to the physiological principles governing the innervation of the heart from the cord and medulla oblongata.

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[1] Cf. *Erb*, Arch. f. klin. Med. I. p. 185. 1865.—*Leyden*, Klinik, etc. I. p. 421.

The *reaction of the pupil* has not been fully studied; and it seldom gives us an unqualified indication. A striking contraction may occur, as well as unilateral or bilateral dilatation. It will, however, not always be easy to decide whether paralysis or irritation of the oculo-pupillary fibres in the cervical cord is the cause of these disturbances, and whether the oculomotorius is not also involved.

The *cerebral symptoms* which occur at times during spinal meningitis and regularly in the cerebro-spinal form—delirium, coma, general convulsions, epileptiform attacks, trismus, grinding of the teeth, disturbances of the organs of sense, spasm and paralysis of single cerebral nerves, vomiting, sleeplessness, loss of speech, giddiness, etc.—are merely enumerated here, as they have been fully described and explained under Epidemic Cerebro-spinal Meningitis (Vol. II.), and Cerebral Meningitis (Vol. XII.).

The *fever* of sporadic spinal meningitis has been but little studied. The temperature seems to be very irregular; in the beginning it is usually high, but at the later period considerable variations occur, which may continue during early convalescence. In fatal cases a rise of temperature is often observed during the agony. As regards the fever of the epidemic form, see the treatise by von Ziemssen in the second volume of this work.

Pathological *eruptions of the skin* (herpes, roseola, petechiæ, erythema, urticaria, erysipelas, etc.) seem not to be constant or particularly significant, except in epidemics; we therefore refer to the place where the latter are described.

The *general nutrition* usually suffers greatly; the patient emaciates quickly and to a great extent, owing to the severity and duration of the fever, the deficient supply of food, the great physical suffering, and the loss of sleep. In severe and protracted cases, emaciation may become extreme.

As a matter of course, not all the cases of spinal meningitis are accompanied by all the above symptoms. In given cases, the appearance of the disease may differ greatly. The leadng symptoms are more or less distinct in all cases, but the accession of the other symptoms, which are not invariably found, may give rise to many varieties of the disease, which cannot possibly be enumerated here. Let it suffice to state that age and individual constitution may produce considerable differences in the course of the symptoms, and that the nature of the cause usually influences the form of the disease; that essential or accidental complications with inflammation of the membranes of the brain, inflammation and other diseases of the internal organs, may greatly modify the character of the disease; and that, finally, the portion of the spinal cord which is attacked has a great influence upon the symptoms.

It is with regard to this latter point that we would briefly mention the chief signs for locating the disease in the lumbar, dorsal, or cervical region.

If the *lumbar region* is the part chiefly attacked, we shall find pain in the loins and sacrum, stiffness of the lower part of the spine, pain radiating to the hypogastrium and the lower extremities, spasm and paralysis limited to these parts, severe urinary difficulties, etc.

If the *dorsal region* is also involved, the pain reaches a higher point of the trunk, pain and stiffness of the back go up as high as the shoulder, disturbances of respiration, præcordial anxiety, etc., are perceived, while the symptoms in the lower extremities continue.

If the process extends to the *cervical region*, the characteristic symptoms of stiffness of the back of the neck, excentric phenomena [*i. e.*, pain, etc., of a central origin] extending to the upper extremities, severe difficulty of breathing and swallowing, anomalies of the cardiac action, pupillary symptoms, etc., are observed.

Finally, if the inflammation extends to the *medulla oblongata* and to the *base of the cranium*, then, in addition to the chief spinal symptoms, those of a cerebral nature become more and more prominent; vomiting, headache, delirium, paralysis of ocular muscles, trismus, disturbances of respiration and speech are observed, and impart to the disease a very characteristic appearance.

Course, duration, termination.—In describing these, we must make a division into groups.

In the severest cases death occurs early. In epidemics it has occurred within a few hours (meningitis cerebro-spinalis siderans), but more usually is postponed for a few days; the violence of the symptoms increases from hour to hour, tetanic spasms interrupt the constant rigidity of the muscles, severe disturbances of respiration and circulation occur, comatose symptoms appear, and death follows amid profound collapse, often preceded by a great rise of temperature, and, in the last hours, a general relaxation of the muscles.

In less violent cases the duration may be two or three weeks; the severity of the symptoms fluctuates, but in general those which import danger increase, the patient's forces sink, and at last the serious symptoms above enumerated appear, and lead to death in like manner.

There are other cases which have a very protracted course. The fever and the acute symptoms diminish, but there is no sign of recovery; the most important symptoms persist and increase, giving rise to the chronic form of the disease. This form may either take the ordinary course of chronic spinal meningitis, or it may be aggravated by a deep affection of the cord, and the patient dies with symptoms of chronic spinal paralysis, often after many months.

As to *recovery*, that is often hoped for in vain. In the most favorable cases it may occur very soon; the threatening symptoms grow less so in a day or two, soon disappearing, or if returning, it is in a milder form and for a short time. This rapid convalescence is often introduced by critical symptoms—profuse sweating, bleeding from the nose, or hemorrhoidal or menstrual bleeding, abundant discharge of urine, etc.; and cases have been reported where patients have been able to go about their business in a week or two. But the recovery usually lasts longer; the convalescence drags on for weeks and months, the pains and paralytic symptoms disappear by degrees, the strength improves slowly, the patient has first to use crutches, and often lingers for months in an invalid condition, from which he finally recovers completely after several courses of treatment. Such are the cases in which inflammatory products, exudations, adhesions, etc., remain to retard recovery.

There are, of course, a few cases in which incurable traces of mischief remain, after the general health has been fully restored; this constitutes an *imperfect recovery*. The patient is well, with the exception of some paresis or paralysis of certain groups of muscles, partial atrophy, local anæsthesia, permanent stiffness of the back, etc. A marked tendency to relapses, of various degrees of severity, often persists for a time.

Diagnosis.

The symptoms, when fully developed, form a very characteristic group, not easy to be mistaken. The chief diagnostic difficulty arises when we have to separate this group from among more complicated symptoms, or to distinguish it from related diseases.

The general marks of the disease are *fever, pain and stiffness in the back, stiffness of the back of the neck, muscular spasms, hyperæsthesia and paræsthesia of the skin, pains in the limbs,* retention of the fæces and urine, dyspnœa, and, *in the later stages, paralysis.* Where all or most of these symptoms are present, it will be quite easy to form a diagnosis.

We shall very often have to put the question whether an existent cerebral meningitis is complicated with the spinal affection. In the cerebro-spinal form, the cerebral symptoms constitute the most prominent feature, while the presence of spinal inflammation is indicated by pain in the back and loins, stiffness of the nucha (which doubtless depends on an implication of the cervical medulla), stiffness of the back, hyperæsthesia and pain in the extremities, particularly the inferior.

The diseases most readily confounded with spinal meningitis are *acute myelitis* and *tetanus*. The diagnosis from acute myelitis may be hard to make in many cases, especially as (in our belief) the two diseases are very often united, and myelitic symptoms are often of more prominence than those of the other disease. But a little attention will enable us to decide the case on one or the other side with very great probability. In acute myelitis the pains in the back and limbs, especially the excentric pains in the limbs, are very much in the background; the stiffness of the back and neck are absent; hyperæsthesia is by no means prominent, and the limbs are not rendered immovable by pain and muscular tension. But in myelitis actual paralysis is very early a prominent symptom; it occurs much more quickly and completely than in meningitis, and in the sensory sphere it is quite prominent in the form of early and severe anæsthesia. To this is soon added paralysis of the bladder and rectum, often acute bed-sore, and much increase of reflex action. The fever is not, however, so high. From these symptoms it will be easy to recognize myelitis if present, either alone or as complicating meningitis.

From *tetanus*, the anatomical basis of which was often supposed to consist of acute meningitis, the latter can usually be distinguished with ease and certainty. Apart from the causal elements, which often much assist in making the diagnosis, the following criteria must be taken into account. Tetanus is a non-febrile affection—in the commencement, at least, it is always such; it begins with trismus, with scarcely an exception, while in meningitis this symptom is postponed to the later stages: there are never any signs of cerebral implication; the peculiar expression due to rigidity of the facial muscles (Koehler, Koenig) is especially characteristic, but does not belong to meningitis; in tetanus hyperæsthesia of the skin is absent, but the reflex excitability is increased to a degree which very seldom occurs in meningitis; the spasms are much more violent and severe in tetanus; very severe dyspnœa and trouble in swallowing occur early, while in meningitis they accompany only the affection of the cervical region and the base of the brain, which is always denoted by marked disturbances of the cerebral nerves, changes in the pupils, etc., that do not occur in tetanus.

It will hardly be necessary to give the points of diagnosis between spinal meningitis and acute *febrile rheumatism of the muscles of the back*, which is characterized by its light and favorable course, the local painfulness of the muscles, absence of pain in the limbs, of hyperæsthesia of the skin, paralyses, etc.

Diseases of internal organs, as, for instance, inflammation of the lungs and pleura, heart, œsophagus, abdominal viscera, etc., cannot possibly be confounded with spinal meningitis except in persons with marked spinal irritation, in whom all febrile diseases are associated with pain in the back, tenderness of the vertebræ, etc. This confusion may easily be avoided by careful attention and physical examination.

Among the forms of spinal meningitis, the *tuberculous* is the only one deserving of careful attention, as its diagnosis is known to involve grave consequences in respect to prognosis and treatment. As it is probably always associated with tubercular basilar meningitis, we may refer to Vol. XII., where are given the points which need attention in distinguishing this form from the other forms of cerebral meningitis. A bad constitution, scrofulosis, tuberculosis, slow development of the symptoms, moderate and irregular fever, retarded pulse, the cerebral symptoms, etc., will guide the diagnosis. Ophthalmoscopic examination, by showing tubercles in the choroid, will perhaps often decide the point.

Prognosis.

According to the form and causes of spinal meningitis, the constitution of the person attacked, the complications, etc., the prognosis will vary extremely. Only the strictest weighing of the case can make clear the leading points, which can only be given in outline in this place.

Cases which begin and continue with sudden violence (foudroyant) are absolutely unfavorable. The tuberculous form is equally so. Those caused by deep bed-sores or severe vertebral lesions are very unfavorable. The rheumatic cases and those due to simple traumatic lesion are more favorable, and so are many cases of epidemic origin.

The progress is influenced *for the worse* by the following circumstances: a very youthful or very advanced age; bad constitution, anæmia, the previous occurrence of severe disease, etc.; by the height to which the disease ascends in the spine, towards the brain; by early symptoms of paralysis, signs of general loss of strength, high fever, continually rising temperature and in-

[illegible] which [illegible]

[illegible] will [illegible]

[illegible] carefully [illegible] with [illegible]

Treatment

[illegible] *prophylactic* measures [illegible] cerebro-spinal meningitis. The precautions to be taken [illegible] are given in [illegible]

[illegible] we have little to hope from [illegible] of foreign bodies, the treatm treat. The severity and threatening nature of the symptoms usually tempt to energetic measures; and, in fact, much has always been done and many things recommended.

Let us keep in mind, above all, that the treatment should be determined upon with careful regard to the individual circumstances, the amount of strength, the causes, the prominent symptoms, and that these ought to be our chief guide in choosing remedies.

We shall, in the first place, always find reason for *energetic antiphlogistic* measures, among which *bloodletting* and *cold* are the chief. General bleeding, venesection, will be used only in the rarest cases, namely, in very robust, plethoric patients, and when the initial symptoms are very intense. *Local bloodletting* will generally suffice; it must be free and frequent. Cups and leeches on the vertebral column are the best, in number dependent on the seat and extension of the disease, the patient's age and constitution. In many cases blood may also be drawn from the anus and vagina.

The application of *cold* along the spinal column must in all cases be attempted, and, if possible, carried out with great thoroughness. Unfortunately, it is often hard to fix the ice-bags on the spine, on account of the patient's restlessness, and yet cold applications and wraps, irrigation and affusion of the back cannot properly replace the bags.

To this add a *vigorous derivation to the intestine or skin.* For the former purpose, drastic purges may be advised (the best is calomel with jalap), or strong saline purges, "aqua laxativa," etc.; of course with allowance for individual constitutions. For derivation to the skin, the best and most successful method consists in the *repeated application of large blisters* along the spinal column. In the milder cases, frictions with pustulating ointment and painting with tincture of iodine along the vertebral column, hot mustard foot-baths, sinapisms to the back and calves or thighs, etc., are sufficient. The white-hot iron seems not to act especially well; it might at most be tried in desperate cases of disease in the cervical region.

Mercury has always been recommended as an antiphlogistic from the earliest times; the most usual methods are the rubbing in of mercurial ointment upon the back or extremities, in the quantity of from one to four grammes [from fifteen grains to a drachm] daily, and the internal exhibition of moderate doses of calomel (0.15–0.25 [from two and a quarter to four grains], two or three times a day); the usual precautions against salivation being of course employed. We have no certain proof of the efficacy of this method. Among the internal remedies, tartar emetic was formerly much given, but is now for the most part abandoned. It is for the future to determine whether *ergotin* deserves the recommendation given by Hammond on account of its action on the vessels.

The entire *surroundings of the patient* must be arranged in conformity with the idea of antiphlogistic treatment; a quiet, airy, moderately warmed room; absolute rest in bed, best on the side or face, avoiding the back as much as possible; prevention of all noise and excitement; avoidance of bodily movement and exertion; for nourishment, a fluid, easily digestible diet, at first cooling, but soon to be made tonic and roborant; for drink, water, lemonade, juices of fruit, mild acid drinks, but no spirituous drink, no coffee or tea—such are the chief things to be attended to.

But this is far from exhausting our therapeutic aims, for there remain very important *symptomatic indications* to be fulfilled, which usually become imperative, owing to the severity of the patient's sufferings.

In the first place, *sedatives* are required, in order to relieve pain, sleeplessness, hyperæsthesia. The *opiates* are universally recommended for this purpose above all other remedies, and they have been sufficiently tested in the great epidemics of cerebro-spinal meningitis; large doses of opium, subcutaneous injection of morphia. With this may be mentioned chloral hydrate, and in some cases the inhalation of chloroform.

May not *belladonna* deserve the preference over opium in such cases? Belladonna is stated to contract the vessels of the cord, and it also possesses narcotic virtues. Favorable sedative results are also to be expected with certainty from the use of bromide of potassium. All these remedies seem especially efficacious when given directly after bleeding.

Baths, especially lukewarm, protracted full baths, occupy

d place as sedatives. If such are used, we may dis-
h cold affusion of the head and back, unless the latter
for by special indications—violent cerebral symptoms,
collapse, etc. *Moist packing* of the whole body often
d brings sleep.

yperæsthesia and pain, muscular tension and spasm,
ls of *external remedies* have been made; little is to be
of them; the most likely to succeed are frictions with
chloroform liniment with infused oil of hyoscyamus,
r applications.

e *fever*, we shall seldom have to take special measures;
ry, we should employ the regular methods of large
uinia and cool baths.

eakness of the heart and *threatened collapse*, the usual
are to be used when required, just as in other inflam-
fections. For *disturbances of respiration* we can do
unless we succeed in directly checking the inflamma-
e cervical region.

alescence has begun quickly and favorably, no further
is usually required, except care of the diet and the
cautions against overexertion. To provide against re-
e wearing for a long time of a vesicating plaster (of one-
sual strength) upon the back has been recommended.

the affection has only assumed the chronic form, it is
bove all, to assist the resorption and withdrawal of the
, for which purpose iodine seems most suitable; to be
rnally in the form of ointment, internally in large doses
of potassium, to be continued for a considerable time.
lief of this stage we must also take into account warm
rmal and brine baths, suitable water-cures, etc., accord-
rules given in the following section on Chronic Menin-

sidua and sequelæ (palsy, atrophy, anæsthesia, weak-
e bladder, etc.) should be treated upon general prin-
stly with baths and electricity. Compare the sections
c Meningitis and Myelitis.

b. Leptomeningitis Spinalis Chronica.

By this term we understand a *non-febrile inflammatio* *the soft membranes of the cord, slow in development and co* or one which has become sluggish after having run an a course. The anatomical changes which characterize this f are usually slight, rarely well-marked. The symptoms, at often very trifling, afterwards assume a more severe chara and may develop by degrees with very destructive effect. C nic spinal meningitis is often the point of origin for chroni flammatory processes in the cord.

Etiology.

The causes of this disease are quite obscure in many resp the disease is very often unrecognized or disregarded, symptoms disappear amid the crowd of symptoms of severe plicating processes.

The disease very often originates from the acute for has, therefore, the same cause. All possible debilitating ences, bad nourishment, abuse of tobacco, etc., are said to the occurrence of this transformation, and, in general, to p a certain predisposition to chronic spinal meningitis.

In general, the same causes which produce the acut may give rise directly to the chronic, provided that they a intense, but perhaps more continuous or more frequen peated.

Many cases are directly traceable to *cold;* the occupa damp dwellings, working in the wet and cold, bivouack bad weather (hence rather common among officers in the Braun), are those most usually spoken of.

Traumatic lesions of moderate severity often lead to c meningitis. Especially, simple shocks, falls upon the s back, contusion of the spine, slight railway accidents a like may by degrees lead to inflammation.

Chronic inflammatory processes or neoplastic proces *neighboring parts* often pass to the spinal membranes ; th ries of the vertebræ, chronic periostitis of the same, carcinoma, d other new formations of the vertebræ or the spinal mem-nes, etc. This connection is especially important in most of e *chronic diseases of the cord;* in chronic myelitis, sclerosis, ophy, and gray degeneration of the cord nothing is more com-n than extension of the chronic inflammation to the soft mem-nes of the spine. Here are to be included the syphilitic and rous affections which often occur in the vertebral canal, the ecific products of which are usually surrounded by more or s extensive chronic myelitis. Bruberger once found an exqui-e syphilitic spinal meningitis of the cervical part of the cord connection with syphilitic basilar meningitis.

How far *suppressed excretions* (*e. g.*, suppression of the hem-hoidal or menstrual discharge, or of perspiration of the feet, e disappearance of chronic eruptions, etc.) may act as causes spinal meningitis, we dare not decide.

The *misuse of alcohol*, however, seems to be certainly a very tive cause (Huss). *Excess of bodily effort* and *sexual excesses* y be considered rather as predisposing than as direct causes.

Koehler lays stress upon chronic disease of the heart and ngs, disease of the liver, and all sorts of circumstances which y give rise to impediments in the vertebral veins, as regular uses of chronic and slow inflammations of the coats of the rd, but he seems not to have paid enough attention to distin-ishing the simple hyperæmia of blocked circulation, with nsudation, from actual inflammation.

Pathological Anatomy.

The morbid appearances are in most cases quite constant, owing, however, some differences in the intensity and extent the process.

Besides a more or less distinct hyperæmia, the principal ana-nical characteristics of chronic meningitis spinalis consist in acity and thickening of the pia and arachnoid, close aggluti-tion and adhesion between these and the dura, unusually firm herence of the pia to the cord, and an abundance of spinal id.

The hyperæmia is mainly of a venous character; the small veins and capillaries are dilated, the color is rather a dark red, more or less diffuse.

The thickening with connective tissue may be very great, so that the soft membranes assume an opaque tendinous appearance, and unite in forming one uniformly hard membrane. The latter may be pigmented in spots, sprinkled with small extravasations of blood and spots of pigment, and is often connected with the dura by means of more or less extensive false membranes. Jaccoud found in an interesting case great fibrous plates in the arachnoid along the region of the nerve-roots, almost continuous in the cervical and lumbar cord, affecting chiefly the anterior, less the posterior roots, and leading to atrophy. It is more common to find a deposition of thin, small, more or less numerous plates of lime upon the arachnoid, which especially in the lumbar region, are often found without any other marked sign of inflammation.

An abundant secretion of spinal fluid is almost always found, seemingly in much increased quantity. Many cases, formerly described under the term hydrorrhachis, evidently come under this class. The serum is often clear and of the usual consistency, but is oftener turbid, flocky, tinged with blood, or mixed with an abundant fibrinous exudation. Stokes found an abundant purulent exudation in a case which had been unattended with fever.

The *dura mater* often shares in the inflammatory process to a corresponding extent, being thickened, opaque, sometimes granular, covered with growths of connective tissue and adhesions (cf. also what was said under Pachymeningitis Interna, pp. 221 and 222).

The *cord itself* is implicated (myelomeningitis) in most cases, though in various degrees and to a various extent. Often only the processes of the pia mater which enter the cord appear thickened and swollen; but more frequently there is a more or less extended sclerosis (so-called) of the cord, in various forms. Such a sclerosis is sometimes annular in shape, beneath the pia, sometimes it occurs as longitudinal bands in certain columns of the cord, sometimes is disseminated in spots, and sometimes it is a chronic myelitis, occupying the entire transverse section, and ding to a greater or less distance longitudinally. We often riginating in such seats of disease, degeneration ascending posterior columns, and descending in the lateral columns, eaching to a great distance. The result of this process e a considerable atrophy and diminution of the diameter entire cord.

e *nerve-roots* are usually atrophied, pale, gray, degenerated; re lost to the eye amidst the thickened and opaque mem- of the spinal cord, and suffer more or less radical changes, rtionate to the intensity and duration of the disease.

e following additional affections of a secondary nature are : atrophy and degeneration of peripheral nerves and mus- ed-sores in all stages and situations, chronic cystitis, etc., s accidental complications, disease of any of the internal s.

Symptoms.

is subject is not quite clear. The disease has not been suffi- y studied, and, besides, is almost always complicated with other affection.

general the symptoms must be the same as those of the form, except that they are much slower and less violent in opment, are without fever, and often are not prominent for g time.

hen the disease develops from the acute form, the violent toms abate, the fever disappears, but a portion of the symp- —of the pain and stiffness, the weakness and abnormal sen- ns—remains for a longer time, and, gradually developing, to an unfortunate condition: it has become chronic menin-

many cases repeated attacks of the subacute form recur; the disease becomes firmly rooted, the intervals between the ks diminish, and the disease assumes a regular chronic form. most cases, however, the disease first appears in the chronic ; the beginning is quite latent, being, at least, not observed e patient, or not considered of any special importance.

he commencement is marked by occasional *abnormal sensa-*

tions in the lower limbs, gradually increasing *pain*, and some *stiffness of the back*. The pain of the back increases, but is usually not very severe, being often described as merely a sense of drawing and pressure in the back, a feeling of weight; it is not usually increased by pressure on the spinous processes or the dorsal muscles, but usually is made worse by movements of the vertebral column. A certain *stiffness of the nape of the neck* is not usual in the early stages.

The excentric symptoms in the trunk and limbs which follow are striking and important. Corresponding with the seat of the disease the annoying *sensation of a girdle* is often felt, and in the same region shooting and boring pains may occur, especially if provoked by movements. In the limbs a *feeling of great heaviness* early appears; in the skin the patients speak of all kinds of *paræsthesiæ*, often of a very singular character—tingling, cold, formication, and the like; these are very often accompanied by tearing or shooting pains, either confined to the region of some nerve-trunk, or else changing from place to place. These pains are exasperated by movement, and not seldom by change of weather, dampness and fog, snow-fall, or a low barometer. Finally, various degrees of *cutaneous hyperæsthesia*, not so marked as in the acute form, have been observed not infrequently.

All these excentric symptoms are confined to the distribution of the nerves whose roots originate in the part chiefly affected. They may, therefore, be most marked either in the upper or in the lower extremities, but the latter is the more usual case.

Symptoms of *motor irritation* are of subordinate importance in chronic meningitis, but are not usually absent. A degree of *stiffness of the back*, and sometimes of the neck, is almost an invariable symptom, and may in some cases become severe. Trembling of the extremities, twitching of certain muscles, sudden starting of the body, involuntary drawing up or extension of the limbs are not rare.

At almost any period in the subsequent development of the disease symptoms of increasing *weakness*, going on to full *paralysis*, become prominent. The heaviness and weakness of the limbs become greater, the patient loses more and more of his control over the extremities, symptoms of sensory paresis, of vesical weakness, of disturbed rectal function occur, and *paraplegia* develops, by degrees increasing in degree and extent.

This paraplegia, though it varies much in different cases, is seldom complete, but usually comprises only a very severe paresis; a certain fluctuation in the intensity of the paresis seems to be rather characteristic; the patient can perform this or that movement better on one day, worse on the next; it is thought that this fluctuation is connected with variations in the amount of fluid exudation in the spinal canal, or the fulness of the circulation; if the spinal fluid is excessive, the paralysis increases when the patient stands, because the lower parts of the cord are more compressed, while, on the contrary, passive congestion may cause it to increase while the patient is lying on his back, and when standing or walking he is better. These two factors, therefore, act in contrary directions.

Severe anæsthesia is rare; there is usually only a slight dulling of the sensations of the skin, limited to the soles, feet, and lower part of the legs. These disturbances are always accompanied by marked paræsthesiæ, not seldom mixed with hyperæsthesia; but the latter is usually not marked, though some reported cases give the impression that it may become very severe.

The associated paralysis of the sphincters increases, and in severe cases there may also be a strongly marked atrophy of the muscles, with loss of electrical excitability.

The disturbances of sensibility increase, the reflex function becomes extinct, bed-sores and cystitis appear, and the scene is closed by marasmus.

Disturbances of any of the internal organs (of respiration, circulation, or digestion) are very common, and originate in the same way as in the acute form, although different from it in degree.

The pathogenesis of the symptoms is about the same as in the acute form; perhaps the cord is still more likely to be affected than in the latter. We can refer upon this point to what has been said under Acute Spinal Meningitis.

Course, Duration, Result.—This disease is always slow and chronic, extending to months and years, often many years. The symptoms often fluctuate considerably; intercurrent, acute exacerbations are not rare.

Some of the cases *recover;* these are the lightest, and those which have early been taken in hand. The return to health is always very slow and gradual, often occurs by successive steps, and is often interrupted by relapses; the sensory disturbances are usually the first to disappear, and the motor remain the longest. Even when recovery is complete the patient usually remains feeble for a long time, with a tendency to relapse.

The *cure* is often *incomplete.* The improvement reaches a certain point, all symptoms of the active inflammatory process disappear, but residua and sequelæ remain, doubtless due to the relics of exudations, compression of the roots by adhesions and thickening, cicatricial sclerosis in the cord, etc. In this category may be included partial or complete paralysis of single muscles or extremities, with or without atrophy, circumscribed anæsthesia, weakness of the bladder, etc.

The cure is often interrupted by repeated relapses.

In a great proportion of the cases chronic meningitis leads directly to *death.* The processes and occurrences which may bring this to pass are many; they usually include the symptoms of grave spinal paralysis—paraplegia, vesical palsy, cystitis, bed-sores, with consecutive anæmia and hydræmia, and, at last, general marasmus. In other cases, the sad termination is brought about by the extension of the process to the cervical region, causing progressive difficulty in breathing and deglutition, secondary pneumonia, etc. In yet other cases, life i speedily brought to a close by the sudden starting into activity of an acute purulent meningitis. There are many other complications and accidents which may hasten the fatal termination o chronic meningitis.

Diagnosis.

Chronic spinal meningitis is often hard to recognize, because the symptoms remain for a long time extremely slight, and, taken as a whole, present an incomplete representation of the disease, or are obscured by complications.

When the whole of the symptoms above mentioned are present, we shall not hesitate long about the diagnosis.

The only difficulty consists in distinguishing between the different forms of *chronic myelitis*, and this difficulty is the greater, as the two diseases are so often combined with one another. The following guide is offered: Pain and stiffness in the back, general pain in the extremities, extensive symptoms referring to the roots, a slight degree of paralysis, equably distributed, and change in intensity dependent on change of position of body, are symptoms which speak strongly in favor of meningitis. The absence of increased tendinous reflex actions, of obstinate contractures, of painful muscular jerkings, may be interpreted in the same sense. Myelitis must be thought of, when severe paralysis and anæsthesia are present, the pains are slight, the tendinous reflex actions are exaggerated, and considerable contracture, etc., is present.

If there is palsy with great atrophy, without any disturbance of sensibility or pain, our first thought must be of myelitis of the anterior gray substance.

From tabes dorsalis, as implying the symptoms of gray degeneration of the posterior columns, chronic spinal meningitis is very easy to distinguish; the points to notice are the characteristic lancinating pains, ataxia, disturbances of muscular sensibility, etc. But it should not be forgotten that both diseases are very often combined, producing a mixed group of symptoms.

In determining the nature of the disease which causes the spinal meningitis, we are guided by general considerations. The diagnosis of the location in the lumbar, dorsal, or cervical region has already been mentioned.

Prognosis.

The disease is in general a grave one; a cure is hard to obtain, especially in rather old and tedious cases. But improvement and cure have occurred, even in seemingly hopeless cases, so that the prognosis is not absolutely bad, even in severe cases.

We may always remember that the chronic inflammation of the spinal membranes need not be a continuous and limitless process; that it is very susceptible of arrest and repair; that the possibility of repair depends essentially upon the organization, calcification, retraction, which may have taken place in the products of inflammation, but that, even after these have taken place, others may often be expected in the course of time, which give rise to considerable improvement of function.

The prognosis may be made by taking such facts into account, and with them the patient's age, constitution, and powers of resistance, state of nutrition, the causes, and the possibility of removing them, the anatomical changes already developed, the intensity of the symptoms, the results of treatment, etc. We must, however, avoid indulging in too great hopes of curing the disease.

Treatment.

The *causal indication* is the first to be attended to. To avoid needless repetition, we would refer to the given list of causes, the proper remedies for which are easily inferred; and we will in this place only emphasize the necessity of carefully treating the *acute* form. We ought always to try to cure it completely, should observe the strictest watch during convalescence, should not let the patient return too soon to the exertions of his calling, should guard him from exposure to cold and other injurious things; by these precautions many cases of chronic spinal meningitis might be prevented.

In the treatment of the disease when developed, antiphlogosis is to be used very sparingly. We shall seldom effect much by bloodletting, energetic derivation to the intestine, etc., in such a chronic disease as the present. Yet there are cases in which these remedies deserve a trial. In robust, well-nourished persons, when the symptoms are rather decided, the pain of the back severe, etc., it will be suitable to apply every week or two from ten to fourteen moist cups along the spine; for feeble patients the application will be restricted to dry cups once or twice a week. Similar principles and special indications (as habitual constipation, hemorrhoids, etc.) will guide us in the use of purgatives.

Derivation to the skin is praised by almost all writers. Noth- seems more suitable for such cases than the repeated appli- tion of large blisters to the back. Brown-Séquard advises to this every fortnight. The mild derivatives (sinapisms, pus- lating ointments, friction with oil of turpentine and croton l, painting with iodine, etc.) may be used in light cases and r the sake of change. The moxa or white-hot iron (burning in reaks along the spine) will be used only in severe and desperate ses.

Of the drugs, the most justly celebrated is iodide of potas- um; it is given for a considerable time in the usual doses. It ill be best to abstain from mercury in these chronic cases, nless syphilis exists. Nothing remarkable is to be expected om ergotin and belladonna. If we have cause to suspect the istence of an abundant serous exudation, we may employ diu- ics.

In many cases *warmth* seems extremely useful; warm gar- nts, furs, and the like, warm wraps, frictions with warm oil, ., are praised.

Upon this fact seems to depend in part the unquestionable antage of *baths*, which have effected many notorious cures in onic spinal meningitis. Warm baths of all sorts, indifferent brine-baths, gaseous brine-baths and chalybeate-baths, etc., y here be used. Braun, who has examined this point with e, states the rule for using them as follows: the baths should general be protracted; the more indifferent the spring, the ger may be the duration; the more abounding in salts and bonic acid, the more should the bath be shortened. The tem- ature should at first be very carefully regulated; if myelitic plications exist, the higher degrees should be avoided, but in e meningitis they seem the best borne, which may account that dangerous credit which many warm springs have ac- red in spinal paralysis. An enlightened diagnosis should be oked in support of such treatment.

In very obstinate cases we may try vigorous *cold-water* treat- nt, especially the wet pack, alternate cold and warm *douches* the back, *moor-baths*, and hot *sand-baths*.

Of the effect of the *galvanic current* in chronic spinal meningitis, we do not yet possess full information. It is extremely probable, *a priori*, that the catalytic effect of the current will be strikingly useful in this disease. An observation by Hitzig[1] seems to confirm this most clearly; the result was obtained by using descending stabile currents. My own experience in the matter is also very favorable, but my observations are too few in number to render a final decision possible. A trial of galvanic treatment (chiefly by stabile currents to the spine, with successive action of both poles) is certainly always indicated, and may very well be conjoined with the use of baths.

Much can also be done to relieve *symptoms*. For pain, the usual sedatives; for paralyses, anæsthesia, atrophy, etc., electricity; for weakness of the bladder, ergot, nux vomica, electricity; for anæmia and cachexia, tonics, iron, quinia, strengthening diet, a little wine, etc. The *diet* must be mostly tonic, adapted to promote and hasten the change of tissue. The *habits* are to be governed by the patient's condition and circumstances, and may be regulated by the general directions given at page 196 et seq.

In all cases, after a cure has been attained, it is absolutely necessary to maintain a long watch over the health and habits in all respects. For a subsequent treatment, many cases will be benefited by electricity, cold-water cures, a mountain climate, and the milder sea-baths.

5. *Tumors of the Spinal Membranes.*

Ollivier, loc. cit. 3d ed. p. 517.—*Cruveilhier*, Anatomie pathol. livraison. XXXII. pl. 1. XXXV. pl. VI.—*Hasse*, loc. cit. 2. Aufl. p. 731.—*Rosenthal*, loc. cit. 2. Aufl. p. 346.—*Hammond*, loc. cit. 3d edition. p. 517.—*Leyden*, loc. cit. I. p. 443.—*Virchow*, Geschwülste. I. pp. 386, 423, 514; II. pp. 92, 120, 345, 354, 461.—*Charcot*, Leçons sur les mal. du syst. nerv. II. Sér. II. fasc. Paris. 1873. —*Jaccoud*, Les paraplégies et l'ataxie du mouv. Paris. 1864. p. 236.—*Brown-Séquard*, Lectures on Paralysis of the Lower Extremities, etc. 1861. p. 92.

Athol Johnson, Fatty Tumour Connected with the Interior, etc. Brit. Med. Journ. 1857.—*Virchow*, Bösartige, zum Theil in der Form des Neuroms auftretende Fettgeschwülste. Virch. Arch. 1857. XI. p. 281.—*Traube*, Fünf Fälle von

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[1] Virchow's Archiv. 1867. Bd. XL.

Rückenmarkskrankheiten. Charité-Annalen. IX. 1861. (Gesamm. Abhandl. II. b. p. 994.)—*Whipham*, Tumour of the Spinal Dura Mater, Resembling Psammoma, etc. Trans. Path. Soc. XXIV. 1873. p. 15.—*Benj. Bell*, Tumour of the Pia Mater, etc. (fibro-nucleated growth). Edinb. Med. Journ. Oct., 1857. p. 331.—*Loewenfeld*, Faserig. Sarkom an d. Wurz. der zwei ersten Sacralnerven links. Wiener med. Presse. 1873. No. 31.—*L. Benjamin*, Neurom innerhalb der Rückenmarkshäute. Virch. Arch. 1857. XI. p. 87.—*Seitz*, Pseudoplasma medull. spin. Deutsche Klinik. 1853. No. 37.—*Charcot*, Hémiparaplégie déterminée par une tumeur, etc. Arch. de Phys. 1869. II. p. 291.—*Baierlacher*, Zur Symptomatologie der Geschwülste am R.-M. Deutsche Klinik. 1860. No. 31.—*Meschede*, Sarkom am R.-M. Ibid. 1873. No. 32.—*Th. Simon*, Tumor im Sack der Dura spinal., die Cauda comprim., etc. Arch. f. Psych. u. Nervenkr. V. p. 114. 1874.—*Simon*, Paraplegia dolorosa. Berl. klin. Wochenschr. 1870. Nos. 35 and 36.—*Davaine*, Traité des entozoaires, etc. Paris. 1860. p. 666.—*Bartels*, Echinoc. innerhalb des Sacks der Dura spin. Deutsches Archiv. f. klin. Med. V. p. 108. 1869.—*Béhier*, Compress. de la moëlle épin. par un kyste hydatique. Arch. gén. Mars. 1875. p. 340.—*Westphal*, Cysticerken des Gehirns und R.-M. Berl. klin. Wochenschr. 1865. No. 43.

Of the new formations within the spinal canal, the most mportant and frequent are those proceeding from the spinal eninges.

They mostly originate in the dura, and develop upon its outer r inner surface; many new formations, however, spring from he arachnoid or pia, and remain limited to these membranes. The latter are not always the point of origin; neoplasms arising rom the neighboring parts often extend to the membranes, involve them in a secondary way, and then produce effects like hose of primary meningeal tumors.

It results from the narrowness of the space within the canal hat the tumors usually found are of inconsiderable size, but hat they very soon begin to awaken the most active disturbnces through irritation and compression of important intraspinal tissues.

Most of the tumors reach only the length of two to four centinetres, rarely that of eight to ten, within the canal; their thickess is from one to three centimetres. Of course, secondary umors, and such as send out branches through natural or artiicial foramina in the spinal column, may reach a much larger size.

The form is usually oval, that of an olive, or something so;

the rate and direction of their growth, and hence also the general character of the symptoms, depend chiefly upon the nature of the tumor. It is said to have been observed that such tumors assume a more vigorous growth during pregnancy.

In enumerating meningeal tumors, we shall for practical reasons not limit ourselves to new formations in the stricter sense, but shall add many things which have the same clinical significance, as inflammatory new formations in the shape of tumors, animal parasites in the canal, etc.

Pathological Anatomy.

The exact histological diagnosis of many intra-spinal tumors is far from satisfactory. It is especially hard to make out in older observations to which of the present categories of new formations we ought to assign them. The observations of the last ten years, though not very numerous, show the existence of the following forms of tumors in the spinal meninges :

Fibroma and *fibro-sarcoma.*—Usually small oval tumors, three to five centimetres long, two to four thick, springing from the dura or the pia, and situated now within, now without the sac of the dura. They consist of connective tissue, with more or less abundant cells, spindle-cells, round cells (transition to sarcoma).

Sarcoma.—Occurring in all possible forms, as hard and soft, fibrous, or cellular ; often with formation of cysts, cysto-sarcoma (observations of Baierlacher, Leyden, and others). Originates more rarely from the dura, more frequently from the soft membranes ; is more usually of a longish shape ; not seldom lobular, with a nodulated surface ; considerable vascularity, and the usual histological characteristics of sarcomatous new formation.[1]

Myxoma has been found by Virchow, Traube, and others in the spinal meninges. It originates almost exclusively from the arachnoid or the pia, and is a soft, juicy, lobulated tumor of moderate size and pale color. It may be pure ; more frequently it is a mixed product, a lipomatous or sarcomatous myxoma, etc.

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[1] See *Virchow,* Geschwülste. II.

To the same series of new formations belongs *psammoma*, found by Whipham, Cayley, Charcot, Bouchard, and others—a sarcoma with granular concretions of lime imbedded in it. Usually a small roundish or olive-shaped, smooth or lobed tumor, in most cases originating from the soft membranes.

Lipomata have repeatedly been found in the vertebral canal, either caused by proliferation of the perimeningeal fatty tissue (Athol Johnson, Obré, Virchow), when it is situated outside of the sac of the dura, or originating from the soft membranes, and situated within the sac of the dura.

An *enchondroma* as big as a hazel-nut, firmly adherent to the dura and the connected vertebra, has once been found by Virchow, and declared to be probably congenital.

Osteoma, the formation of new bone, in the form of the so-called cartilaginous disks, is extremely common in the arachnoid, but in this form cannot be considered a tumor, and has no clinical importance. Ossification of the dura also occurs only in the diffuse form.

Multiple fibrous *melanoma* has been seen in the spinal canal by Virchow and Sander.

The name of *neuroma* has been given to many new formations occurring in the spinal canal, on the nerve-roots, especially on the cauda equina (Benjamin, Virchow). These are mostly the so-called false neuromata, and occur either singly or in numbers.[1]

Carcinoma proper seems very rarely to spring from the spinal membranes; I, at least, have had knowledge of no unquestioned case of primary carcinoma of this region, excepting the older and less reliable observations of "fungous" or "cancerous" growths. The tumors are almost always secondarily developed by extension from the vertebræ or other neighboring parts, or by metastasis from other organs. Such secondary carcinomata not seldom appear in the vertebræ, in consequence of primary cancer of the breast.

We have spoken under meningitis of the formation of miliary *tubercle* in the membranes of the cord.

With these are associated the *tumors which originate in*

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[1] See Vol. XI. p. 599 et seq.

inflammatory, hemorrhagic, and other processes in the spinal membranes or the neighboring parts. Such are peripachymeningitic exudations, with or without caries of the vertebræ, those hard, circumscribed growths, of a purulent or caseous nature, of which we have before spoken, and which are so common in Pott's disease; also the greenish-yellow, bacony, scrofulous exudations between the dura and the vertebral column; and the hæmatoma of the dura mater, originating in pachymeningitis interna hæmorrhagica.

The *syphilomata* which are occasionally found in the spinal membranes are of a somewhat similar nature (Wilks, Virchow). They have been little studied; they usually consist of gummata of the dura or the pia.

Finally, we have to speak of the parasitic new growths which occur (though rarely) in the spinal canal.

Cysticercus cellulosæ has been found once by Westphal in the sac of the dura; there were numerous cysts in the lumbar portion, some free in the fluid, some firmly enclosed in the meshes of the arachnoid; a few also in the thoracic and cervical portions. Numerous cysts in the brain. Only one of them contained a head. Clinical symptoms referred to the spine had been present.

Echinococcus has been found more frequently (thirteen times in all) by Davaine, Cruveilhier, Lebert, Foerster, Rosenthal, Bartels, etc. The development of most of the cysts took place externally to the dura, and often led to considerable tumors outside of the vertebral canal. In only two cases (Esquirol and Bartels) did they develop within the sac of the dura. They differ in size, and present the usual characteristics of colonies of echinococcus.

With this we close our enumeration—perhaps an imperfect one.

Respecting the *seat* of meningeal tumors, we need not add much. They may occur at any part of the canal, may compress the cord in front, behind, or at the side, may include various numbers of pairs of nerve-roots, etc. Each case will differ in these respects, but one thing is tolerably important and characteristic, namely, that the disease always occupies a quite limited and definite locality.

But, for the purpose of understanding the clinical symptoms and the entire course of the disease, it is of much importance to attend to the *consecutive changes* which always, though in varying degree, accompany the formation of tumors in the spinal canal.

The *nerve-roots* in the region of the tumor are sometimes found swollen, reddened, softened by inflammation, sometimes thin, flat, grayish and translucent, atrophied and degenerated, according to the duration and extent of the process.

The *cord* itself always experiences some degree of compression, which may change it to a flat, ribbon-shaped string, or, at least, produces a local indentation, more or less deep. Simple atrophy of the portion compressed is seldom the only change. It is much more common to find at the compressed point marked inflammatory symptoms (*myelitis from compression*), which can be followed but a short distance upward, and often a good way downward. The medulla is in a state of whitish or reddish softening, is full of small hemorrhages, and exhibits under the microscope many granular corpuscles amid the *débris* of nerve-elements. This change continues to a variable distance downwards in the white substance, and more especially in the gray also. Cruveilhier once found the whole peripheral portion of the cord in a state of purulent degeneration.

The examination of the hardened cord in these cases regularly shows secondary degeneration, ascending in the posterior columns, and descending in the postero-lateral (see farther on, II., No. 19). Simon has also found this ascending degeneration in a tumor of the cauda equina.

In the *membranes of the cord* we almost invariably find signs of chronic inflammation (thickening, opacity, pigmentation, hyperæmia, etc.) of various extent. In correspondence with this, an increase of the spinal fluid (hydrorrhachis) is pretty constant.

In the *peripheral nerves and muscles, degenerative atrophy* is not rare; usually in those nerve-districts whose roots are either directly included in the new formation, or are directly connected with degenerated portions of the gray substance.

Such further alterations as are found on the bodies of those dead of meningeal tumors—bed-sores, cystitis, extreme marasmus, changes of internal organs, etc.—will be described hereafter

Etiology.

The causes of meningeal tumors are usually obscure. It seems to be established that *surgical injuries* are the most likely to produce them; cases have been observed where the first symptoms occurred after a fall or blow upon the back, or spine, etc.

Exposure to cold has been assigned as the cause of the disease in a number of cases, and, as it seems, with sufficient reason.

It is remarkable how often it is recorded that the first symptoms appeared during *childbed*, shortly after delivery.

Observations by Cruveilhier and Kohts render it probable that excessive mental excitement, violent *fright*, may give the impulse to the formation of meningeal tumors.

Finally, *vertebral disease*, the *tuberculous and scrofulous diathesis*, and *syphilis*, are among the causes of meningeal tumors, as follows from the preceding enumeration of the forms. The entrance of animal parasites is effected in the usual way by swallowing the eggs, or the proglottides of the various species of tape-worm.

It is seen that these scanty facts leave a large part of the etiology of meningeal tumors in obscurity.

Symptoms.

The *general outline of the symptoms* usually caused by meningeal tumors may be drawn in a few words.

If the disease begins (as it often does) quite in a latent and insidious way, the development of the tumor is usually first indicated by the presence of *pain* in certain nerve-districts, which increases in severity. *Abnormal sensations* in the trunk (feeling of a girdle) and the extremities, *paræsthesiæ, anæsthesia, partial paralyses*, are associated with the above—all at first localized in the same nerve-districts. *Painful stiffness* of a definite portion of the spine indicates more directly the seat of disease.

After a period of various length, often after years, *paraplegia* follows, and increases; it begins as a progressive paresis, often unilateral, like Brown-Séquard's palsy, but usually making rapid progress across the body, and ending with absolute sensory and motor paralysis. The disease rarely remains stationary at a point of moderate severity, but usually goes on to extremes; and after great suffering, attended by all the horrible circumstances of the severest spinal paralysis, palsy of the bladder and rectum, cystitis, extensive bed-sores, general marasmus, etc., the patient is brought to a miserable end.

Although the individual features of this sketch are by no means characteristic of meningeal tumors when taken separately, yet, when considered as a whole, they often enable us to recognize and to localize the disease with some accuracy.

Upon closer consideration we find two groups of symptoms, distinguishable both by their succession in time and by their pathogenetic significance. These are:

1. *Symptoms of local irritation and compression of the nerve-roots and the membranes first involved in the tumor.*

2. *Symptoms of irritation and compression of the cord itself, and of consecutive myelitis* (*myelitis by compression*).

Those of the first group are the earliest, and often precede the paraplegia by months and years; their character of course differs in each case according to the location, direction of growth, and rate of growth of the tumor; so that no exhaustive statement can be made. But the numerous varieties may be inferred from the general scheme. All these symptoms arise from irritation or compression of the nerve-roots, from consecutive irritation of the meninges, and, to some extent no doubt, from commencing irritation of the cord itself.

Violent *pains* are next observed, the lancinating, tearing, boring character of which proves their excentric origin. They may remain confined to a single point, or attack a single nerve-trunk; hence, according to the seat of the disease, they either surround the trunk like a girdle at various levels, or invade the upper or lower extremities, of one side or both. They may extend suddenly or by degrees to neighboring nerve-districts, are often increased by movement of the spinal column, and seem to be made worse by sudden changes of weather (Bell). These symptoms are so intimately associated with those of compression of the cord by tumors, that Cruveilhier distinguished *paraplegia dolorosa*, due to compression of the cord, from paraplegia non dolorosa, caused by primary disease of the cord.

Paræsthesiæ also occur, corresponding to the extent of the pains—the sensations of tingling, formication, numbness, deadness, etc., either in the form of a girdle or limited to certain regions of the extremities.

If motor roots are first exposed to the influence of the tumor, separate *muscular twitchings, spasms*, may appear at the beginning.

These symptoms of irritation are almost always accompanied by distinct, sometimes very active *pain in the back*, localized in the neighborhood of the tumor, and usually accompanied by a local *stiffness of the spine.* Leyden points out the fact that the movement of the spinal column is often difficult and painful in a certain direction, because this motion brings a greater pressure upon the tumor.

In the further course of the disease *symptoms of paralysis* appear sooner or later, corresponding to the locality of the disease; circumscript anæsthesia, often coinciding with the distribution of nerves which are especially painful (anæsthesia dolorosa), local paralyses and pareses of the corresponding muscular groups, atrophy, etc., compose an extremely complicated and varied group of initial symptoms.

It needs only to be indicated how various are the phenomena of this first group in connection with different seats of disease; how when the cervical region is the part attacked, for example, one upper extremity may first be seized by pains, paræsthesiæ, partial palsy and atrophy, before the symptoms of compression of the cord appear; how, when it is the dorsal region, the disease will be introduced by intercostal neuralgias, paroxysms of visceral pain, zoster, etc.; how, finally, when the lumbar region is involved, all the above disorders may appear, now in the district of the sacral, now in that of the lumbar plexus, and put on a great diversity of form. Numerous and instructive examples of this are upon record.

When the symptoms of the first group have existed for an uncertain length of time (weeks, months, often several years), they are succeeded by *symptoms of the second group*, derived from the continuous pressure upon the cord, and usually from myelitis also (which hardly ever fails to be present); these become more and more distinct, and change the scene in a very disagreeable way.

The development may be rapid or slow; it often occurs with almost suddenness in a few hours, and in such case is nearly always due to secondary myelitis, as it is seldom we can suppose so rapid an increase of the pressure of a tumor. The compression is often limited at first to one lateral half of the cord, which may give rise to the characteristic symptoms of Brown-Séquard's hemiplegic lesion (paralysis of the side of compression, anæsthesia on the opposite side; see the section on Unilateral Lesion, *II.*, No. 14.) for a longer or shorter time. Or the compression may occur on the anterior or the posterior surface of the cord, the result of which is, that in the former case the motor phenomena, in the latter the sensory, not only preponderate, but often are exclusively present for a time.

After a variable time, the palsy invades all the paths in the portion of the cord adjoining the tumor, and we have the symptoms of extreme compression of the cord. We shall describe them fully in the section on "Compression of the Cord" (see farther on, II., No. 5), and will therefore mention here only the most important in order to complete the description, referring for all details to that section.

First of all there is severe *paraplegia;* motility and sensibility more or less completely paralyzed, up to the level corresponding to the seat of the tumor; the limitation of these paralytic symptoms upwards is variously strict. The bladder is paralyzed; at first the symptoms of retention are prominent, and later those of incontinence, with continuous dribbling. The sphincter ani is also paralyzed.

Violent pains in the parts below the seat of lesion usually follow. Although the doctrines of physiology are opposed to the supposition that such excentric pains could originate in compression or irritation of the cord itself, yet many observations (as those of Whipham, Leyden, Brown-Séquard) show that violent pain of that kind in the legs may be caused by tumors seated in the upper dorsal or the cervical region. It is certain that in most cases the palsied parts are at times the seat of very severe pains, perhaps due to secondary myelitis.

In the *motor apparatus* also, the *symptoms of irritation* do not fail to accompany those of complete palsy; muscular twitchings, spasms, and contractions—at first temporary, then permanent—occur, while the muscles, at first quite relaxed, become by degrees tense and rigid (secondary degeneration of the lateral columns).

In many cases there is a remarkable *increase of reflex actions.* Slight cutaneous irritation produces free and vigorous muscular contractions, powerful flexion or extension, active clonic tremor, more marked in the lower extremities, especially when the tumor is seated at an elevated point. If, however, the gray substance is compressed by the tumor (when located in the lumbar region), the reflex actions are wholly wanting. The same is the case when the gray substance is deprived of its functions by secondary descending myelitis. For this reason, in the later stages, we often see the *reflex activity diminish* and disappear, when previously it had been exaggerated. The *tendinous reflex* acts also seem considerably increased.

The nutrition of the muscles is affected in nearly the same way; at first it is well kept up, but afterwards severe *atrophy* appears. The *electrical reaction* behaves in the same way; at first well preserved, it may afterwards sink and disappear.

Paralysis of the bladder may lead, after a while, to *cystitis,* with ammoniacal decomposition of the urine and the presence of abundant pus. The absolute immobility of the patient, who is almost always forced to retain the dorsal decubitus, and the frequent befouling of his person with urine and fæces, etc., give rise to *gangrenous bed-sores* on the sacrum and buttocks, trochanters, heels, etc., which often make unchecked progress and produce the most shocking destruction.

Chills, with very high temperature—a more or less continuous *fever*—appear. This, and the loss of fluids caused by the suppurating sores, and the loss of sleep and appetite from continual pain and abnormal sensation, continually increase the patient's anæmia nd cachexia, and cause a marasmus which of itself is sufficient o produce the fatal result. Death usually occurs in a state of opor, with a rising temperature, and often after a very pro-onged agony. In other cases the patient's sufferings are brought o an end by bronchial catarrh or pneumonia, or an acute menin-itis caused by bed-sores, or other diseases.

According to the seat of the tumor, the rapidity of its growth, he patient's power of resistance, the sequence of these symp-oms may be rapid or slow. If the seat is high in the cervical egion the course is usually very rapid, paralysis of the respira-ory nerves producing early death by suffocation.

Course, Duration, Termination.—The course is usually slow nd insidious, especially at the beginning, and the first period of he disease may last several years. With the occurrence of araplegia the second period commences; this often comes on ther quickly, in a few days or one or two weeks, but may quire a much longer time. In a few cases it is effected quite ddenly, and is then usually dependent on myelitis—as in a se by Ollivier, in which advanced softening was found after ath.

After paraplegia has appeared the progress is usually more pid, but even at this point years may pass before death occurs. his, of course, depends on the rate of growth of the tumor and e height at which it is situated. Temporary improvement is casionally mentioned, and great fluctuations in the severity of mptoms may be observed; such are usually referred, for the ost part, to the state of the compressive myelitis, but in part so to changes in the volume of the tumor caused by variation the amount of blood contained, processes of softening, changes the direction of growth, etc.

The entire *duration* cannot always be easily determined, ving to the uncertainty as to the date of commencement. Some ses have terminated fatally in eight or ten months, while others n on for one, three, or five years; and cases of much longer ration—extending to fifteen years—have been observed.

The *termination* is almost always in death in the manner escribed. A permanently stationary condition or improvement, r cure, is rarely seen. Yet the *possibility* of such an event, at least for certain forms, cannot be denied. In syphilomata and scrofulous tumors, in inflammatory new formations, in cysticerci (by shrinking or calcification), it may certainly be admitted, and in the case of others may be held an open point. It will certainly be hard to prove such an event, both because the diagnosis during life is very obscure, and because in cases which have recovered, it will seldom happen that the spinal canal will be opened post-mortem.

Diagnosis.

The diagnosis of a meningeal tumor is sometimes quite easy, but usually very difficult, and for a long time uncertain. When the symptoms as above given develop quickly and promptly, and with their full characteristics, or if the disease has passed into the second stage, there is usually no great difficulty. But previous to this point, years of uncertainty and guess-work may pass. And, on the other hand, in cases with slightly marked symptoms, the disease may possess an unlucky resemblance to a great variety of circumscript diseases of the cord, rendering a distinction impossible.

The diagnosis chiefly depends on the demonstration of a *slowly developed compression* of the cord (see farther on, II., section 5), which has been preceded by the signs of a *circumscribed* irritation or compression of certain portions of the roots. It should be particularly noticed that the symptoms point only to a progression of the paralyzing lesion *transversely* to the cord, while an extension in the *longitudinal* direction is not perceptible, at least *not upwards*, while a descent of the process of myelitis is not rarely observed. This, however, does not make much change in the symptoms.

When we have made out the existence of a tumor with some degree of certainty, we may consider ourselves prepared to attack the second and more difficult question as to the *nature of this tumor*. In settling this question, we are often entirely deprived of guiding-points—especially as the position of the cord renders a direct examination in most cases quite impossible. In many cases something may be found to base a diagnosis upon, and in these, after a careful estimation of all circumstances, we shall be obliged to follow general pathological principles.

We shall infer, for instance, a peripachymeningitic exudation if Pott's disease or a marked scrofula exists; a carcinoma, if there is cancer of the vertebræ or primary cancer of some other part; a syphiloma, if syphilitic infection can be demonstrated; an echinococcus, if the parasite has been found in other organs, or tumors containing the cysts are demonstrated near the spine; a neuroma, if neuromata are found in peripheral nerves, etc. In most cases, however, we shall be forced to confine ourselves to guesses.

It is easier, in most cases, to define the *precise seat of the tumor*, or, if there are several of them, at least that of the uppermost. This is done by following the rules we have repeatedly given, and which will be stated more precisely below, depending on the local distribution of the symptoms of irritation and paralysis.

We may here add a word upon the *tumors of the cauda equina*, which usually proceed from the meninges, and have, in every respect, a great resemblance to those which are situated higher and affect the cord proper. They are hard to distinguish from the latter, but may be in many cases, perhaps, if it is borne in mind that tumors of the cauda produce *exclusively nerve-root symptoms*, and that the signs of compression of the cord, of secondary myelitis, etc., are absent. The higher the tumor, the nearer it approaches the lumbar portion of the cord, the harder will it be to draw the distinction. In respect to tumors seated lower, the following points may be attended to: the seat of the pains (which in such cases often attain enormous violence) is strictly localized in certain nerve-districts; all nerves leaving the spinal canal above the tumor are free; thus, in myxo-sarcoma telangiectodes of the cauda, I observed the pain strictly limited to the district of the sciatic, while the crural and the dorsal nerves were perfectly free; constant violent pain in the sacrum. If palsy occurs, the reflex actions necessarily cease at once. Spasms are seldom observed; more frequently contractures. Atrophy of the muscles occurs rather frequently. The palsy and anæsthesia, by their localization, often give us the opportunity of fixing the upper limit of the lesion. Increase of the reflex acts and marked tendinous reflexions, do not occur. Paraplegia, palsy of the bladder, bed-sores, etc., may develop exactly as in tumors occupying a higher seat; but the symptoms of paralysis do not seem to belong necessarily to the disease, as is shown in my case (just mentioned), which terminated fatally before paralysis or anæsthesia occurred.

Prognosis.

It follows from what was said of the course, that the prognosis of meningeal tumors is very bad in almost all cases. If there are actual neoplastic formations, the prognosis is absolutely bad. In the most favorable case, the disease may cease for a limited time to make progress, or death may be delayed. The more quickly the symptoms develop, the worse for the patient; and the worst is in carcinoma. In some other tumors, in the inflammatory, scrofulous, hemorrhagic, and syphilitic forms, the prognosis is more favorable, and is decided upon general principles.

If paraplegia has become complete, the case is usually hopeless. The early or late occurrence of the fatal termination then depends on the rapidity and intensity with which cystitis and bed-sores are developed. The prognosis of each case will depend on the circumstances and constitution of the patient, the possibility of sufficient attention to his wants, on the special prominence of certain symptoms or complications, etc.

Treatment.

This presents as little encouragement as the prognosis. For the disease properly considered (if there exists a new formation in the strict sense) as good as nothing can be done. Some success is to be hoped for in inflammatory, syphilitic, scrofulous forms. We should in general attempt to fulfil the causal indication, as far as possible.

The object of removing or diminishing the size of the tumor has been sought in a variety of ways, but mostly without success. All sorts of local derivatives have been tried, from painting with tincture of iodine and blistering, to the moxa and the white-hot iron. Internally, it will be proper to try iodide of potassium and ioduretted mineral waters, mercury, arsenic, etc. Some improvement in symptoms has been observed from the use of warm springs and brine-baths. As long as such temporary improvement (often lasting for quite a while) can be effected, and while the diagnosis remains uncertain, we shall always be tempted to make new trials.

If the diagnosis and the seat of the tumor are quite certain, we may perhaps consider the question of trepanning the vertebral column; but it is seldom that we shall find firm ground for undertaking such an heroic operation. And yet the success of this attempt is by no means beyond the limits of possibility, especially if the tumor lies *outside* of the sac of the dura, upon its posterior surface. If we are forced to open the dura, the danger is much increased. At all events, the prognosis of the disease is so desperate that we ought to take the operation into consideration. Echinococcus cysts, growing from the spinal canal, should be opened and evacuated, or extirpated.

In the great majority of cases, the chief object will consist in *general attention to the patient's needs*, and *treatment of the symptoms*. In respect to the former, we have nothing to add to what we said in the general part of this work. Above all, we ought to prevent cystitis and bed-sores if possible, and to keep up the strength of the patient.

As regards symptoms, the pains should be the first and constant object of treatment; they often bid defiance to all remedies, and enormous doses of morphine are usually required to render the patient's life tolerable. The entire range of narcotic and anti-neuralgic remedies must be tried. For the paralytic symptoms nothing can be done directly. Cystitis and bed-sores are to be treated upon general principles.

Addendum.

Anatomical Changes in the Spinal Membranes, without Clinical Significance.

Various changes in the spinal membranes are known to pathological anatomy which seem to produce no symptoms during life; sometimes they are senile changes, sometimes slight incidental inflammatory or degenerative disturbances, which remain without symptoms as long as they affect the membranes alone and do not involve the roots or the cord. These matters are about equal in importance to pleuritic adhesions in the respiratory apparatus.

The physician, however, ought to know them, in order not to interpret things which are usually innocent as the cause of symptoms observed during life. They are therefore briefly enumerated in this place.

1. *Bony and cartilaginous disks in the arachnoid* are very often found. These are small, roundish or angular plates, flat, from six to fifteen millimetres in diameter, somewhat thicker in the middle, and sharp on the edges. Their number in the arachnoid varies; they are especially frequent in the lumbar part and on the posterior surface of the cord, often composing a literal mosaic. While Ollivier[1] considered them as purely cartilaginous, Virchow[2] has shown that they consist of young bony tissue, and have a structure which agrees for the most part with that of the cartilage of bone; a striped basement substance in strata, inclosing star-shaped bodies, and passing by calcification directly into bone-tissue. Their outer surface is smooth, their inner more rough and jagged, so that they feel like a cat's tongue.

In most cases they give rise to no symptoms whatever. In advanced age they are almost always present. They are often found in young persons who have exhibited no spinal symptoms. They certainly point to an irritative condition of the meninges, especially if they exist in large numbers. They are probably referable to slight, often-repeated irritation. The connection with epilepsy, formerly imagined to exist (Esquirol, Ollivier), is at all events very doubtful.

2. *Diffuse ossification of the spinal membranes* occurs now and then, but seems destitute of clinical significance. Diffuse ossifications have been described in the dura (Andral, Virchow), and small osteophytic elevations are also seen in the same membrane; the deposition of brain sand [corpp. arenosa] in the exudations with thickening of the dura (pachymeningitis arenosa[3]) may perhaps be included here.

3. The *pigmentation of the pia* may often reach so high a degree as to become pathological. Pigmented corpuscles of connective tissue are often found in the pia soon after puberty, especially in the cervical region. In marked cases this may give rise to a diffuse, slightly brownish, smoky gray or blackish coloration.[4] A gradual transition from this state to actual melanoma has been observed. The simple pigmentation is destitute of pathological importance, and has no demonstrable connection with inflammation, or with epilepsy, as was formerly supposed.

4. *Small tumors of all sorts* may exist innocuously. Fibromas, cysts, melanomas, neuromas, etc., from the size of a hemp-seed to that of a pea, have been repeatedly observed in the cauda and the membranes without accompanying symptoms. This is very easily understood.

5. An *increased amount of spinal fluid* must not be regarded as a necessary cause of decided disturbances of function.

Such an increase is most frequent (hydrorrhachis externa[5]) in the meshes of the arachnoid in connection with atrophy of the cord. As long as this has the character of normal spinal fluid, its increase is not of any great importance. But if it is turbid, of a reddish or whitish tinge, contains numerous cell-elements, blood-corpuscles, etc., its increase is due to an increase of irritation or to a considerable blocking of the circulation of the spinal membranes, and then represents simply a portion of the general morbid process. But even then there is no reason for referring the cause of severe symptoms exclusively to the pressure of the increased volume of spinal fluid, as was formerly often done.

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[1] Loc. cit. 3. Aufl. II. p. 466 seqq.

[2] Geschwülste. II. p. 92.

[3] *Virchow*, ibidem. II. p. 117.

[4] Ibidem. II. p. 120.

[5] *Virchow*, Geschwülste. I. p. 175.

II. Diseases of the Spinal Cord proper.

Introductory.—Before entering upon the special consideration of these diseases, it will be suitable to give a reason for the order and distribution of the material which we have adopted.

We cannot pretend, in the present state of our knowledge, to make a strictly scientific division of diseases of the spinal cord. To base such a division upon strict principles of pathological anatomy would be completely impossible, as we are in the dark with reference to the significance of many processes, and in many diseases are ignorant of any anatomical basis.

A division by special localities is equally impossible, for in many diseases we are quite ignorant of the exact locality, and in other cases such an arrangement would involve a great deal of repetition.

We must, therefore, resort to such methods as are convenient.

It seemed to us that for practical purposes we ought to present as full an account as possible of the important parts of spinal pathology, without too much detail or repetition. The principle adopted is simple and intelligible, and sufficiently consistent with the rules of logic.

In the *first group*, comprising eleven sections, we shall speak of the processes *which occupy, or may occupy, the entire transverse section of the cord in a diffuse way;* processes which do not, at any rate, either necessarily or regularly, imply a limitation to certain portions of the transverse section. They may extend longitudinally to various distances. They comprise hyperæmia, anæmia, and hemorrhage of the cord, acute severe traumatic lesions, and slow compression of the cord (Nos. 1–5).

Then follow three diseases: concussion of the cord, spinal irritation and spinal nervous debility (Nos. 6–8), in which we are aware of no anatomical changes, but are allowed to suppose at any rate fine disturbances of nutrition, which, more or less diffused in the transverse and longitudinal directions, are certainly not attached to certain parts of the transverse section.

In Nos. 9–11 we shall present the inflammation of the cord (acute and chronic), simple softening, and that peculiar anatomical change commonly known as sclerosis in patches—processes which likewise do *not* derive their characteristic features from their connection with any certain portion of the transverse section.

In the *second group* (Nos. 12–16), we shall treat of those forms of disease, as far as known, which are marked by their *more or less strict localization in certain portions of the transverse section of the cord*, while their extension in the longitudinal direction may greatly vary and change. To this class belong the degenerative processes in the posterior columns, in the lateral columns, and the lesions which are limited to one lateral half of the cord; also the acute and chronic processes confined to the gray anterior cornua.

Under No. 17 we shall describe acute ascending paralysis, a form of disease still perfectly obscure, which, for the better understanding of it, we shall place after the above-named diseases.

The placing of tumors, secondary degenerations, and malformations of the cord (Nos. 18–20) at the end of this series is justified by their less frequent occurrence and importance, and the impossibility of including them directly in either larger group.

Finally, in No. 21, we present a number of scattered facts, which have an undoubted, though in many cases an obscure connection with the pathology of the cord, and which deserve to be collected as material for the further development of the subject. As inducements to further investigations and researches, they may be granted a little space.

1. *Hyperæmia of the Cord.*

We have already spoken of this, as indistinguishable from the same condition of the membranes, either by anatomical or linical tests; and would refer the reader to the complete account iven on page 199 et seq.

2. *Anæmia of the Cord.*

asse, loc. cit. 2. Aufl. p. 652.—*Hammond*, loc. cit. 3d edition. p. 396.—*M. Rosenthal*, l. c. 2. Aufl. p. 290.—*Leyden*, l. c. II. p. 27.—*Jaccoud*, Les paraplégies et l'ataxie du mouvement. Paris, 1864. p. 293 et seq.

Stenon, Element. myologiæ specimen. Flor. 1667.—*Kussmaul* and *Tenner*, Unters. über Ursprung und Wesen der fallsuchtartigen Zuckungen bei Verblutungen, etc. Molesch. Unters. zur Naturl. III. 1857. p. 59.—*Schiffer*, Ueber die Bedeutung des Stenson'schen Versuchs. Centralbl. f. d. med. Wiss. 1869. Nos. 37 and 38.—*Ad. Weil*, Der Stenson'sche Versuch. Diss. Strassburg, 1873.—*Romberg*, Lehrbuch der Nervenkrankh. 2. Aufl. I. 3. p. 2.—*Barth*, Oblitérat. complète de l'aorte. Arch. gén. 1835. VIII. p. 26.—*Gull*, Paraplegia from Obstruction of the Abdom. Aorta. Guy's Hosp. Rep. 3d series. III. p. 311. 1858.—*Cumings*, Paraplegia from Arteritis. Dubl. Quart. Journ. May, 1856.—*Panum*, Zur Lehre von der Embolie. Virch. Arch. XXV. 1862.—*Brown-Séquard*, Lectures on the Diagnosis and Treatment of the Principal Forms of Paralysis of the Lower Extremities. London, 1861.—*Sandras*, Traité des maladies nerveuses. Paris, 1851.—Service of *Grisolle:* Paraplégie après une métrorrhagie considérable. Gaz. des hôp. 1852. No. 108.—*Moutard-Martin*, Parapleg. causées par les hémorrh. utérines ou rectales. Soc. méd. des hôp. 1852. Union méd. 1852.—*Abeille*, Études sur la paraplégie indép. de la myélite. Paris, 1854.—*Van Bervliet*, Observ. de parapleg. chlorotique. Annal. de la soc. méd. de Gand. 1861.—*Mordret*, Traité prat. des affect. nerveuses et chloro-anémiques. Paris, 1861.

Definition.—By anæmia we understand a diminution in the mount of blood contained in the cord; and this may consist of:

a. Diminution or complete suspension of the arterial supply o the cord—ischæmia; or,

b. Deficiency of blood in the cord, owing to *the diminution of he total amount of blood, and bad crasis* (oligæmia, hydræmia, tc.), and usually named anæmia without further definition.

The first form has been experimentally studied, and has been n single cases made the object of clinical study.

The second form is still less distinct clinically, owing to the requent want of prominence of the spinal symptoms, and to imultaneous disturbances, cerebral and otherwise, originating in he anæmia, and obscuring the spinal symptoms.

It is plain that there are many transitional forms between these two, and that a sharp distinction is not always feasible, although we shall attempt to make it in the following presentation. Both forms lead to the same result, although in some cases it may be developed quickly and in others slowly; *the nutrition of the substance of the cord will suffer more or less*, and this will bring about the corresponding disturbances.

Pathogenesis and Etiology.

The conditions which may produce a marked *predisposition* in certain persons to anæmia of the cord are not fully studied. Various points may, however, be mentioned which deserve future attention. For instance, *congenital narrowness of the calibre of the circulation*, shown by Virchow to be so frequent an occurrence in chlorosis; also congenital or acquired *weakness of the heart;* and finally, an *undue excitability of the vaso-motor nerves*, so common in nervous persons, which may sometimes be most marked in the cord. It is a fact which may be connected with these circumstances, that the *female sex* seems specially predisposed to certain forms, at least, of spinal anæmia. Diseases of the vessels of the cord also, which are of quite frequent occurrence, doubtless give rise to a certain predisposition to anæmia. I am inclined to refer to anæmia of the cord those slight attacks of feebleness of the lower extremities which are found in old people with marked atheroma of the arteries.

The *direct causes* of spinal anæmia are better known.

The first group embraces all those causes which produce a *contraction or closure of the afferent arteries of the cord* (paraplégies ischémiques of Jaccoud). Of these we must first name *compression, thrombosis or embolism of the abdominal aorta* above the point of departure of the lumbar arteries. It leads to a severe ischæmia of those segments of the cord which receive branches from the corresponding lumbar and intercostal arteries.

It has very long been known that compression of the abdominal aorta is followed very quickly by paralysis of the posterior half of the body (Stenson's experiment). The paralysis occurs a few moments after the compression begins, and was rred by all the earlier observers to a peripheral disturbance of the nutrition of es and muscles. Kussmaul and Tenner have, however, shown that anæmia of cord leads to palsy much more rapidly than anæmia of the nerves and muscles. iffer has tested the question again, and has decided that anæmia of the cord lf is certainly the immediate cause of the palsy, that the nerves and muscles in case remain excitable for a long time, and that in case of compression at a er point, the paralysis occurs much later. It is also true that when the com- ssion lasts a considerable time, paralysis of the cauda equina, the peripheral es and muscles follows. A. Weil has confirmed Schiffer's statements in all ntial points. Such ischæmic paraplegiæ have been seen to occur in the human ject, in the rare case of thrombosis and embolism of the aorta (Barth, Gull, den, Tutscheck, and others), although it is not always possible to decide whether r origin is spinal or peripheral.

Thrombosis and embolism of given spinal arteries can only d to quite circumscribed ischæmia, owing to the numerous erial supplies which the cord possesses.

This cause has been studied experimentally by Panum, but in man has been accidentally observed, and its pathogenetic import is not yet sufficiently de- l. Leyden has found capillary embolism of the cord in ulcerous endocarditis.

It is still uncertain whether *spasm of the spinal vessels* can duce anæmia of the cord; the fact is not well established, ugh not quite improbable. We may assume that a direct tation of the vaso-motor paths concerned produces this kind ischæmia; but the theory has been more generally accepted t irritations of peripheral organs, due to all sorts of causes, y produce such vascular spasm in the *reflex* way; and this y be the origin of a great many of the so-called "reflex paral- s."

The latter view has been developed into a theory of "reflex paralysis" by wn-Séquard in particular. The peripheral irritation causes a contraction of the al vessels, of various duration, which gives rise to palsy and a more or less per- ent disturbance of nutrition in the cord, as has been observed by Kussmaul and ner, and by Schiffer, to occur after simple compression of the aorta, if contin- for a sufficient time.

Although it is not certain that such a permanent and severe spinal vascular m occurs, as is demanded by Brown-Séquard's theory, and although on the r hand many "reflex paralyses" can be referred to severe disturbances of nutri- , yet a transitory spasm of the spinal arteries is by no means inconceivable.

Vaso-motor spasms of the skin of the extremities may last for hours and days;[1] why not in the cord also? And if such a thing occurs, severe disturbances may occur in the fine and delicate processes of nutrition of the central apparatus.

It is self-evident that *mechanical* pressure upon the cord may produce ischæmia in a corresponding portion; in such cases, however, the symptoms are referable rather to pressure upon the nerve-elements than to compression of the blood-vessels.

The second group of direct causes embraces all such as diminish *the total amount of blood*, or cause a decided *change in its composition*, with a tendency to oligocythæmia, hydræmia and allied disturbances. (A portion of Jaccoud's paraplégies dyscrasiques.)

Upon the whole, the existence of severe spinal symptoms, especially of paralyses, in these conditions is comparatively rare, and the causal relation between the anæmia and the paralysis which follows is not always perfectly clear. It seems at first strange that the lower extremities should be almost exclusively affected; but a closer consideration shows, as Jaccoud has admirably stated, that the fact is probably due to the greater claims usually made upon the activity of the legs at all times, which causes any general weakness to show first in them. It will also remain undecided in many cases whether and to what extent the symptoms of weakness depend on anæmia and disturbances of nutrition in the peripheral nerves and muscles. For this reason cases must be read with some caution.

Several cases are described in which paraplegia followed *great loss of blood*, as in parturition, metrorrhagia, bleeding from the kidneys and intestine, epistaxis, etc. Jaccoud quotes such cases from Grisolle, Moutard-Martin, Abeille, Landry, and others.

A similar effect is produced by *great loss of blood*, *severe acute diseases*, *inanition*, etc., which injure the nutrition of the cord by producing great anæmia.

In *chlorosis*, states of weakness and palsy have been observed and referred to anæmia of the cord; such cases are rather frequent, though not so in comparison with the frequency of chlorosis itself. Jaccoud quotes such from Dusourd, Bervliet, Bouit, Mordret, Landry, and others, and includes the paraplegia pregnancy in the list. It appears that only quite severe cases hlorosis cause such paraplegia.

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[1] See *Nothnagel*, Vasomotorische Neurosen. Deutsches Arch. f. klin. Med. II.

Pathological Anatomy.

Anæmic portions of the cord look pale, bloodless, white; no nts of blood appear on their cut surface, and no full vessels to be seen; the gray substance is strikingly dull in color, and s a little upon the section; the white is often found very soft semifluent, and pushes out above the cut surface. There however, reports of cases in which the substance of the cord found somewhat firmer and more resistent than usual. haps these were different stages of the same change.

'he membranes also appear pale, their vessels partly empty not easy to see.

distinct contrast to this is afforded by those sections of the in which the circulation is retained, which look of a rosy r and are harder in consistence; an increased injection, with avasations of blood, is often observed in the neighborhood of mic portions.

n general anæmia the cord is usually anæmic also.

t is not always easy to avoid confusing these phenomena post-mortem appearances; anæmia will therefore be conred to have existed during life only when all the circumces which produce it in a corpse can be shown to be absent, as a certain position, cadaverous swelling of the medulsubstance, etc.

n thrombosis and embolism of the small spinal vessels it is n possible to find the point of occlusion. Red softening ts in the region supplied by the plugged artery, and in its nity collateral fluxion. This has been found chiefly in ani-. Tuckwell has found similar appearances in man. Leyden rved microscopical embolic foci in ulcerous endocarditis.

f the ischæmia is protracted, secondary changes occur—e and yellow softening of the corresponding portion of the , localized breaking-down, stasis of blood, etc. The minute changes in nutrition which appear in a short time are not accessible to a pathologico-anatomical examination.

Symptoms.

Before describing anæmia of the cord, let us consider such facts as are derived from experimental physiology, and which, at least in the case of the acute ischæmic form, furnish us abundant information.

When the *aorta is compressed*, motor and sensory palsy of the legs immediately occurs, the reflex acts cease, the bladder and rectum seem paralyzed. When the circulation is restored, improvement in these respects is slow in proportion to the duration of the compression.

Precisely the same symptoms occur in man after embolism of the aorta—rapid palsy of the legs, sphincters, reflex function, etc. Gull's case, in which he observed paralysis to occur in a few minutes, is an especially good instance of paraplegia from aortic obstruction. In most other cases of this class it is less easy to affirm that the paralysis originates from spinal anæmia; it is rather probable that the disease has a peripheral origin (see the cases of Romberg, Cumings, Leyden, Tutscheck, et al.).

If the contraction of the aorta develops by slow degrees, th disturbances are of a gradual and less severe nature—slight feeling of numbness and of a limb asleep, weakness of the lower extremities, which are easily fatigued when severe exertions ar made. The symptoms of closure of the aorta become more an more distinct—absence of pulse in the crural arteries, coldne and œdema of the feet, enlargement of collateral arteries, etc.—symptoms which in acute cases appear very quickly.

Here must be included a series of cases which present the symptom of *int mitting lameness* or palsy. In these no change is observed while the patient is rest; but when he takes a brisker walk than usual, it is followed by distinct wea ness or even palsy, which disappears when he takes rest, and again returns wh he renews his muscular efforts. Such symptoms have been observed in horses, a are accounted for by occlusion of the aorta. A similar thing has been seen in m (Charcot, Frerichs—intermittent palsy in *one* lower extremity), the cause of whi is also doubtless referable to the closure of one iliac or the aorta. But these se

only cases of peripheral palsy; the muscles, imperfectly supplied with fresh
d, become incompetent to their duty where severe exertions are demanded,
e they are still able to perform a slighter task.

All that has been said applies only where the ischæmia is sit-
ed in the lumbar region of the cord. We know nothing in
ard to the symptoms of ischæmia of the cervical part. Clo-
e of both vertebral arteries might be followed by ischæmia in
case also; but the derangement of the cerebral functions
those of the medulla oblongata would then probably be so
ere as to mask the spinal symptoms, and death would follow
edily.

The symptoms of *vaso-motor ischæmia* of the cord must be
same, but they can hardly be so severe. We know nothing
th precision in regard to them, excepting those symptoms of
eflex paralysis" which are said by Brown-Séquard to origi-
te in this way. It is said to be characteristic of these paraly-
s that they originate in peripheral irritation, that variations in
e severity of such irritation are followed by corresponding
ctuations in the symptoms of palsy, and that the latter often
appear when the former cease. It is evident how imperfect
his characterization.

Anæmia originating in *thrombosis and embolism of small
eries* probably gives rise to merely local and subordinate
ptoms, about which nothing is known definitely. If large
ts of softening are formed, the symptoms of circumscribed
truction of the cord follow, which will vary somewhat accord-
to the seat of lesion; we shall come back to these in the sec-
on Softening of the Cord, No. 10, farther on.

In the *second group* of cases of spinal anæmia the number of
ptoms of anæmia is so great, involving most of the organs of
body, as to make it hard to sift out those proper to the affec-
.

The most constant seem to be those of *motor weakness*—
kness and great fatigue, which forbids all severe exertions,
ht tremor when even the least muscular work is performed,
in the higher degrees, severe paresis, and at last paralysis.
this usually begins in the lower extremities, and extends
gradually to the trunk and arms.

The *sensibility* is usually intact; but paræsthesia of every sort, pain, and hyperæsthesia or slight anæsthesia occur. The *reflex actions* are often exaggerated; only in the severest cases are they depressed. The *sphincters* do not seem to be usually affected, unless the severest anæmia with full paraplegia has been developed.

At the same time, the most marked symptoms of general anæmia or developed chlorosis exist.

It is said to be a marked characteristic of this form, that the symptoms are improved by continued lying down, which favors the flow of blood to the cord; changes of circulation produce the same effect upon the severity of the symptoms. It is important to note in conclusion that a tonic treatment with iron and stimulants quickly improves such cases.

Hammond has tried to prove that the so-called *spinal irritation* depends on a local hyperæmia of the posterior columns. We shall return to this point in speaking of spinal irritation (see farther on, No. 7).

Course, duration, termination.—The disease may begin rapidly and acutely, as in embolism, severe loss of blood, etc. At other times it is slower and more gradual, as in thrombosis, chlorosis, etc.; the symptoms at first do not appear until certain efforts are made, but become by degrees more distinct and permanent until the disease is fully developed.

In its further course the patient either recovers rapidly, by the establishment of collateral circulation or regeneration of th lost blood, or perhaps by relaxation of a vaso-motor spasm;

Or, after long fluctuation, a slow recovery occurs, especiall when the circulation becomes free after having been disturbe for a sufficient time to leave considerable impairment of nutr tion;

Or, finally, return to a normal state is impossible, the cor softens, producing all the symptoms of severe spinal paralysi in the midst of which death at last occurs.

Of the *duration* of the disease nothing need be said, as it ma vary very greatly according to the cause, the possibilities o repair, the development of secondary changes of nutrition, etc.

Diagnosis.

We are not entitled to infer the definite existence of spinal anæmia from the above symptoms, unless the causes are clear.

The acute ischæmic form often commences quite like a spinal hemorrhage or an acute myelitis; the diagnosis becomes probable only when the aorta can be proved to be closed, or a great loss of blood has recently occurred; and it may be confirmed by the rapid and favorable course of the disease. We have already said that an intermittent character of the paralytic symptoms could not probably be referred directly to anæmia of the cord.

The chronic anæmic forms (dyscrasic) resemble chronic myelitis, or very slow forms of chronic meningitis, etc. If, however, chlorosis or severe general anæmia exists, we shall naturally think first of anæmia of the cord. The fact that the horizontal position relieves the symptoms may perhaps be made of use in the diagnosis; but usually the decisive test will have to be furnished by the result of treatment.

Vaso-motor ischæmia is probably hard to distinguish. Believers in Brown-Séquard's theory of reflex paralysis will refer to it when peripheral irritation exists (diseases of the organs of urination or digestion, or of the uterus, etc.). The idiopathic forms require some further study and proof.

Prognosis.

This depends chiefly on the causes, and on the possibility of removing them. We shall, therefore, have to decide the question on general principles.

Of itself, spinal anæmia is nothing serious. If it has not lasted long, or has never been extreme, the prognosis is quite good; especially so in chlorosis.

But a severe anæmia may badly impair the nutrition of the cord, even when the disease has lasted but a short time, and may cause injuries which require a long time for their repair. Experiments have sufficiently proved this, and we ought, therefore, to be cautious in making the prognosis of such cases.

If it be impossible to restore the circulation, and if softening has once occurred, the prognosis is bad, provided large portions of the cord are affected. If there is softening of small portions of the cord, the prognosis should depend on the size and location of such spots.

Treatment.

Here the first point is the *causal indication.* If we succeed in removing the causes of spinal anæmia, the chances of restoration increase considerably. The reader is, therefore, merely reminded of the treatment of aortic thrombosis and embolism (proper position, stimulation of the function of the heart, etc.), of chlorosis and anæmia (tonics and iron), and of general nervousness; of the removal of sources of peripheral irritation, etc. These measures will generally include the chief part of the treatment.

As direct measures for the relief of anæmia of the cord, we would recommend a *suitable position*, in order to favor the flow of blood to the cord. Brown-Séquard strongly recommends the dorsal decubitus, with raised head, arms, and legs; and this should be maintained during the night, and several hours in the daytime.

Drugs which increase the flow of blood to the cord, especially strychnia, opium, and nitrite of amyl. Brown-Séquard recommends strychnia above all others, and Hammond strongly supports the recommendation; he gives it in increasing doses (0.002-0.015, [$\frac{1}{30}$ to $\frac{1}{5}$ of a grain], three times a day), or, still better, in combination with phosphorus (extract of nux vomica 0.02 [$\frac{1}{3}$ of a grain], phosphide of zinc 0.006 [$\frac{1}{11}$ of a grain]).

Galvanization of the spine, with the object of dilating the vessels of the cord and improving the spinal nutrition. Hammond especially recommends the ascending stabile current.

Application of *warmth* to the back, by means of hot sand-bags, or Chapman's bags filled with hot water. For vaso-motor ischæmia, alternate cold and hot douches are recommended.

We shall also seek to fulfil the *symptomatic indications* (relief of pain, paralysis, disturbances of circulation, etc.) by the usual remedies and methods.

The *diet* and *regimen* must be governed by the existing indications and circumstances.

3. *Hemorrhage in the Substance of the Spinal Cord—Hæmatomyelia (Hæmatomyelitis) — Hæmorrhagia Medullæ Spinalis—Spinal Apoplexy.*

Compare the repeatedly quoted works of *Ollivier* (II. p. 167), *Jaccoud* (p. 251), *Hasse* (p. 667), *Hammond* (p. 440), *M. Rosenthal* (p. 292), and *Leyden* (II. p. 54). Also

E. *Levier*, Beitr. zur Pathologie der Rückenmarksapoplexie. Diss. Bern, 1864 (containing all the older cases).—*Hayem*, Des hémorrhag. intra-rhachidiennes. Paris, 1872 (list of cases complete to that date).—*Breschet*, Hématomyélie. Arch. de méd. XXV. 1831.—*Grisolle*, Rev. hebdom. des progr. des sci. méd. 1836. No. 3.—*Monod*, De quelques maladies de la moëlle épin. Bull. de la Soc. anat. 1846. No. 18.—*Cruveilhier*, Anatom. pathol. livr. III. pl. VI.—*Gendrin*, De l'apoplexie rhachidienne. Gaz. des hôp. 1850. No. 48.—*M. Trier*, Hosp. Meddelelser. Bd. IV. 1852 (quoted in Levier. Schmidt's Jahrb. Bd. 78. p. 293). —*Lebeau*, Cas d'hématomyélite. Arch. belg. de méd. milit. Janv. 1855.—*Barat-Dulaurier*, Sur les hémorrh. de la moëlle. Thèse. Paris, 1859.—*Duriau*, De l'apoplexie de la moëlle épin. Union méd. 1859. Nos. 20-25.—*Brown-Séquard*, Paralysis of the Lower Extrem. p. 86. 1861.—*Colin*, Hémorrh. de la moëlle. Soc. méd. des hôp. 1862.—*Mouton*, Consid. sur l'hémorrh. rhachid. Thèse. Strasb. 1867.—*Schuetzenberger*, Apoplexie spinale. Gaz. méd. de Strasb. 1868. No. 5.—*Koster*, De pathogenie der apoplex. medull. spin. Nederl. Arch. voor Geneesk. IV. p. 426. 1870.—*Gorsse*, De l'hémorrh. intramédull. etc. Thèse. Strasb. 1870.—*C. O. Joerg*, Fall von Spinalapoplexie. Arch. d. Heilk. XI. p. 526. 1870.—*Bourneville*, Hémorrh. de la moëlle ép. Gaz. méd. de Paris. 1871. No. 40.—*Liouville*, Hématomyélie avec anévrysmes. Soc. de Biolog. 1872.—*Erb*, Ueber acute Spinallähmung. Arch. für Psychiatrie u. Nervenkrankh. V. 1875. Beob. 5. p. 779.—*H. Eichhorst*, Beitr. zur Lehre von der Apoplexie in die Rückenmarkssubstanz. Charité-Annalen I. (1874) p. 192. Berlin, 1876.—*E. Goltdammer*, Zur Lehre von der Spinalapoplexie. Virch. Arch. Bd. 66. 1876.

Definition.—The above titles include *any kind of extravasaion of blood in the substance of the cord proper.* This is as rare s meningeal hemorrhage, one important reason for which is cerainly the low and comparatively constant pressure of the blood n the small spinal arteries.

The intra-medullary hemorrhages are seated almost excluively in the gray substance; their occurrence in the white substance is rare, and perhaps is never spontaneous and primary. Their extent is usually small, but is often considerable, and may reach to the whole length of the gray axis.

In these diffuse cases, it seems at present quite doubtful (Charcot, Hayem, Koster) whether the affection is a primary idiopathic hemorrhage, and not rather a hemorrhagic myelitis. This is certain, at least, that a great deal has been classed as hæmatomyelia which is not included under spontaneous and primary bleeding; such cases will have to be sifted in future. It is, no doubt, correct that in many cases of hæmatomyelia there is simply a myelitis complicated with hemorrhage (myelitis centralis hæmorrhagica); but this ought by no means to be so extended as (with Hayem) to include *all* bleeding in the substance of the cord, and make it depend on previous myelitis. We are decidedly of the opinion, particularly upon the ground of clinical symptoms, that *primary* hemorrhage of the cord also occurs, though the way for it may often be prepared by the occurrence of slight alterations in the vessels or the cord. A case which seems to us to be very convincing in this regard is the one lately published by Goltdammer.

The chief symptoms, and the course of the disease, are so alike in both forms that we may treat of them together. We shall, however, return to the inflammatory hemorrhages of myelitis at a later point.

Pathogenesis and Etiology.

Our knowledge of *predisposition* to hemorrhage of the cord is very scanty. The small number of cases hitherto reported seem to show that youth and middle age are the most frequently attacked (as the largest number occurred between the ages of ten and forty), in opposition to cerebral apoplexy, the frequency of which increases with advanced age.

Men are attacked much oftener than women, probably on account of their modes of life.

It is not known how far the occurrence of the bleeding is favored by the existence of heart disease (hypertrophy of the left ventricle), spinal curvature, etc.

It is, however, certain that *disease of the spinal vessels* (thickening, fatty deposits, increase of the nuclei in the walls, aneurysmal dilatation—Liouville) constitutes an important predisposing cause. The same is true of *chronic affections of the cord proper* (chronic myelitis, progressive muscular atrophy, tumors, etc.), which often are brought to a sudden close by hemorrhage. The influence of these circumstances may reach so far as to give rise to apparently spontaneous bleeding, for which reason we shall again speak of them among the direct causes.

Among these *direct causes* we have to name, in the first place, *surgical injuries*. Spinal apoplexy has been known to be caused by a fall or blow upon the back, by vertebral fractures and dislocations, by violent shocks in riding, by a fall down-stairs, etc., without any direct traumatic lesion of the cord.

In the second place, all circumstances which produce a *strong active congestion* of the cord. Such are exposure to cold, sexual excesses and masturbation, extreme exertions of body, and the like. Fluxions originating in the collateral way ought also to be included, as the cases of spinal apoplexy following retention or suppression of the menses (Levier, Schuetzenberger), or suppressed hemorrhoidal bleeding, or those which occur in the neighborhood of inflammatory processes in the vertebræ, the dura, etc.; and with these should be included the spots of red softening which originate in embolism of the spinal arteries. And finally, inflammatory congestion, which so often leads to capillary hemorrhage in acute central myelitis and similar conditions.

Anything which produces a *disproportion between the pressure within and that without the blood-vessels* may give rise to hemorrhage in the cord. When the pressure of the atmosphere is rapidly lessened (in going out of caissons where the air is compressed, used in the construction of bridges, or out of diving-bells), symptoms have been seen which indicated spinal apoplexy, but this has not yet been confirmed by autopsies. A considerable increase of the blood-pressure acts in a similar way; such increase occurs in the case of excessive cardiac action, or in a more passive way it is due to impediments to the circulation in diseases of the heart and lungs, to sudden severe bodily exertion in lifting heavy burdens, in severe spasms, etc.

Another group may be formed of those causes which *lessen the resistance of the walls of the blood-vessels*, and thus give rise to bleeding. Here should be mentioned the aneurysmal dilatations of minute vessels, as found by Griesinger and Liouville; the fatty degeneration, thickening, multiplication of nuclei, etc., in the walls of the small arteries, which are often found in microscopical examination of the diseased cord; perhaps also the chronic processes of softening and inflammation in the cord, and tumors of the cord (especially the soft myxoma and myxosarcoma), in the interior or vicinity of which bleeding so often occurs. We must include here also the bleeding which in rare cases accompanies hemorrhagic affections (scorbutus, hemorrhagic small-pox, etc.), or acute infectious diseases (typhoid, yellow fever, malarial diseases, etc.)

Pathological Anatomy.

The bleeding is mostly confined—in many cases exclusively so—to the gray substance, and in it attains very various dimensions. It may involve the gray cornua or the entire transverse section of the gray substance, and may extend to various distances longitudinally. Bleeding in the white substance is much rarer, and is almost always combined with bleeding in the gray.

Two sorts of extravasation may be distinguished by their visible characteristics, which may coexist, but in origin and appearance are essentially different.

1. The *hemorrhagic (or apoplectic) clot.*—We find a clot of blood, of variable size, as big as a pea, an almond, or at most a nut; often showing through the pia as a bluish lump, while the pia is pressed up and often burst by the pressure, so that blood is found in the subarachnoid space. The clot itself is composed of blackish red blood, coagulated, and sometimes fluid in the centre. The substance of the cord is broken down to a corresponding distance, and forms a sort of ragged wall around the clot. An envelope of white substance commonly surrounds the clot; it is tinged with blood (red or yellowish) to a greater or less distance, so that the boundary between the clot and the sound

tissue is largely obliterated. The clot sends out processes to various distances in the gray substance, and between the bundles of white fibres.

The clot is almost always greater lengthwise of the cord; the globular form is usual only in very small extravasations; considerable portions of the gray columns are commonly affected, producing what is called a tubular hemorrhage. One single clot is what is usually found, but several, or many, sometimes occur. The cervical and upper dorsal region is by far the most frequently affected, though of course not always.

The microscope shows a great number of blood-corpuscles in all stages of decomposition and change; pigment granules and pigment crystals, fibrin, broken-down medullary elements, globules of myelin, and usually granular corpuscles also.

The clot undergoes a series of *further changes* in time; it either thickens and slowly dries up to a crumbly, caseous lump, the origin of which is denoted by its color and the presence of crystals of hæmatoidin, or the process of softening and fluidification occurs, so that at the last a hard capsule of connective tissue is found filled with serous or atheromatous contents. Smaller extravasations may doubtless be absorbed, for the most part, and leave behind only a small cicatrix of connective tissue, colored ochre-yellow by deposits of crystalline pigment.

Secondary disease of the cord is very often found in the neighborhood of the clot. This most frequently consists of softening, which extends to various distances up and down, often to nearly the whole length of the cord. Hemorrhagic softening of the gray substance is especially frequent, by which it is changed into a porridge-like mass, sometimes reddish black, sometimes of a chocolate color, and sometimes of an ochre-yellow (see the drawing in Cruveilhier); in these cases there probably always exists a primary central myelitis. But simple white softening also occurs in the neighborhood of the clot; it is to be recognized by its characteristic appearance to the naked eye, and microscopically by the numerous granular corpuscles, broken-down nerve-fibres and ganglion-cells, the vessels in fatty degeneration, and the proliferation of neuroglia. In older cases we find secondary ascending and descending degeneration of the posterior and lateral columns, presenting the same characteristics as in other limited disease of the cord (Goltdammer).

2. *Hemorrhagic infiltration or softening;* inflammatory hemorrhage. This likewise occurs in the gray substance exclusively, and is either limited to certain cornua or extends over the entire section, but rarely spreads to the white substance. It has been seen with an extent of a few centimetres, and again, reaching the whole length of the cord.

The gray substance is changed to an atheromatous mass, reddish brown, dotted with darker, blackish red points, and small coagula; the blood is intimately mingled with nerve-substance. Around about the place, at various distances, the cord is tinged unevenly of a rusty or ochre color.

The microscope shows essentially the same elements that exist in the clot, but with a greater prevalence of granular corpuscles; traces of growth of connective tissue and of histological changes in the nerve-fibres and ganglion-cells are also found.

The latter can usually be traced in the gray matter far beyond the limits of the hemorrhagic infiltration; they comprise softening, accumulation of granular corpuscles, thickened and swollen axis-cylinders, like strings of beads, enormously swollen ganglion-cells (Charcot), abundant proliferation of connective tissue, excess of blood in the smallest vessels, some of which are enlarged into ampullæ (Liouville), while in other cases their walls are thickened and degenerated—in a word, acute central myelitis.

Of the further changes occurring in such hemorrhagic infiltrations nothing precise is known, for autopsies are usually made at an early period.

Capillary hemorrhages proper—small punctated extravasations, easily recognized—are rather common; they are not specially important, and give rise to no clinical symptoms. They constitute, however, a frequent element in important processes, softening, and the like. In their most developed form they are simply equivalent to hemorrhagic infiltration. Eichhorst has lately examined under the microscope a remarkable case of hæmatomyelia with widely-distributed capillary bleeding, and has given a careful description. We are not able, however, to agree with him in considering this case as a *primary* hemorrhage, as there existed paraplegia, accompanied by fever, *gradually* extending upward, and in a few days leading to death.

Changes in the spinal meninges are not specially essential accompaniments of intra-medullary hemorrhage. There is almost always hyperæmia, corresponding to the seat of hemorrhage; rarely ecchymoses.

The *peripheral nerves and muscles* often undergo a marked degenerative atrophy; this depends on the seat of lesion, and probably on the destruction of the trophic centres of these organs.

The alterations in the *other organs* are the same as in the other forms of severe spinal paralysis (see the chapter on Myelitis).

Symptoms.

Although the number of observations is somewhat small, yet it is possible to draw a tolerably fair outline of spinal apoplexy.

The attack *begins* quite suddenly in many cases, and with fulminant symptoms; the patient is struck with violent pains and sudden paraplegia, and falls without loss of consciousness.

The hemorrhage often occurs in sleep, and the patient awakes paralyzed.

But the disease does not always begin so very suddenly, being often preceded by *premonitions*, which either consist of the symptoms of *spinal congestion* (pain in the back, excentric pain and paræsthesia in the limbs, great weariness and prostration, hyperæsthesia of the skin, etc.), lasting perhaps for days and weeks, or the symptoms of *acute central myelitis* (general *malaise*, fever, violent pains, formication, the sensation of a girdle, of heaviness and numbness, distinct weakness in the extremities, weakness of the bladder, etc.), and these usually last hours or days, until the apoplectic paraplegia appears.

One specially characteristic feature of spinal apoplexy is the way in which *a complete and severe paraplegia will develop in the course of a few minutes or an hour;* this is usually introduced by a violent pain, either localized or extending over the entire spinal column, but usually disappearing after the palsy has become complete.

Such patients are found by the physician with complete and absolute palsy of the legs, or the palsy may extend upwards over the trunk and even to the upper extremities; then the respiratory muscles are palsied, and the patient becomes a picture of helplessness, breathing painfully and imperfectly with the aid of the diaphragm. *The paralyzed muscles are perfectly lax*, offering not the slightest resistance to passive movements.

In rare cases the paralysis is incomplete, some movements being left, or there is paresis only. In one case a single upper extremity has been seen palsied (Bourneville); hemiplegic paralysis is also rare, and always affects the upper extremity more than the lower. All this depends on the position and extent of the hemorrhage.

The motor paralysis is accompanied by *anæsthesia more or less complete* in respect to all possible sensory impressions; this has the same distribution as the motor paralysis. It is obvious that this disturbance may vary in degree, and is subject to exceptions; but a certain amount of anæsthesia is seldom or never absent.

Paralysis of the bladder and rectum is equally regular; at first there is complete retention of the urine, requiring the employment of a catheter, and afterwards various forms of incontinence; the stools are evacuated involuntarily and unperceived.

Marked *vaso-motor* paralysis has been found in well-observed cases. Levier found a rise of temperature in the paralyzed lower half of the body (flexure of the knee), which equalled 0.2—0.5—1.0—2.0° Centigrade as compared with the axillary measurements; a symptom which, if it be of long duration, points not merely to a simple severance of the vaso-motor paths, but also to a destruction of the vaso-motor centres in the cord. Levier found the cutaneous perspiration absent in the paralyzed parts.

The *reflex actions* vary much according to the seat of lesion; they are completely suspended when the gray substance is wholly destroyed to its lowest point; if the seat of the hemorrhage is higher, they may also disappear at the first moment, owing to the shock, but soon return, and may become much exaggerated. In a few cases *priapism* is given as one of the symptoms.

While the lower half of the body is thus severely affected, the upper half may be perfectly normal and healthy, the arms may ct normally, consciousness, intelligence, the functions of the erebral nerves remain quite intact. At the most, slight febrile ymptoms appear during the first few days.

It is somewhat striking that the *symptoms of irritation* are o much in the background. *Pain in the back* seems the most requent, and may be localized or extensive; the spine is little or not at all sensitive to pressure, a high degree of such sensitiveness being probably limited to myelitis.

Although *symptoms of motor irritation*, as twitching and partial spasms, are observed in the first moments of the hemorrhage, yet at a later period they become very inconsiderable, and spasmodic symptoms are almost entirely confined to the non-paralyzed parts, thus marking the progress of the fundamental lesion or the supervention of secondary affections. Paræsthesiæ may be wholly absent in the paralyzed parts; the patients do not feel their limbs, or only feel them as a dead weight; in other cases tingling or similar symptoms are felt in the paralyzed regions.

In the succeeding days and weeks these symptoms increase in a very troublesome way.

The first threatening symptom is usually the rapid appearance and the steady progression of *gangrenous bed-sores* on the sacrum, the trochanters, the heels, and other places exposed to pressure. This untoward event may occur in a few days, often in its most acute form.

The *urinary excretion* is altered, the discharge soon becomes bloody, purulent, albuminous; the severe palsy of the bladder eads directly to alkalescence of the urine, cystitis, and pyelitis with their sequelæ.

It follows, of course, that these severe disturbances are always accompanied by marked *fever*. Chills occur, pyæmic and septicæmic symptoms are developed from the bed-sores, and rapidly consume the patient's strength.

The *paralyzed muscles become atrophic*, sometimes very rapidly; and the atrophy is accompanied by a loss of faradic excitability, or the appearance of the reaction of degeneration in the muscles. A few muscles become rigid or contracted, especially when secondary changes in the cord occur at a late period; spontaneous spasmodic jerkings, with exaggeration of reflex action, usually precede the appearance of these symptoms. But if the lesion is located high, the nutrition of the muscles and their electrical excitability may remain nearly intact, as in the case of Goltdammer.

The *reflex actions disappear* by degrees, often quite rapidly and completely; this is especially the case in central myelitis, when it spreads downward.

It is usually hard to recognize the symptoms of *secondary myelitis;* violent pains, twitching movements and jerks, the formation of contractures—all this often in parts not affected by the palsy—such are the symptoms which belong to this affection.

It is evident that this description applies in full only to the more severe cases, with large effusion, but must undergo various modifications according to the seat, size, and cause of the hemorrhage. It seems superfluous to attempt a full presentation of these; the reader will be able by reflection to see for himself the special symptoms of a small limited hemorrhage; for instance, that in the anterior cornua it will produce mainly symptoms of local paralysis, in the posterior cornua perhaps very inconsiderable symptoms, etc. It should in particular be stated that in many such cases, with small hemorrhage, the symptoms are so indecisive, so destitute of characteristic traits, as to furnish no means whatever of forming a diagnosis of hemorrhage. This is in entire accordance with what we know of spinal pathology.

Hayem gives the name of *chronic* spinal apoplexy to the cases in which the hemorrhage is an accompaniment of existing chronic spinal disease. He quotes the cases of Massot (progressive muscular atrophy), Nonat (chronic central myelitis), Lancereaux (peri-ependymal myelitis). In all these, the symptoms of bleeding were more or less acute in their appearance. In our opinion, a hemorrhage in the cord cannot be chronic. Such cases are simply instances of the supervention of an acute complication (hemorrhage) upon a chronic spinal disease; not at all of a chronic form of spinal hemorrhage.

s to the characteristic symptoms as dependent on the lity of the bleeding, we will content ourselves with a few ls.

f the *lumbar region* is affected, the symptoms of palsy and sthesia are restricted to the lower extremities, bladder, and um; reflex actions are absent; rapid atrophy of the muscles reaction of degeneration, bed-sores at an early period, will ly fail to be present.

f the *thoracic region*, the symptoms extend higher up on the k. The expiratory muscles are palsied, and those which press the abdomen; reflex actions may be retained for a e; atrophy of the muscles is tardy.

If the *cervical region*, all four extremities are affected. A rtion of the inspiratory muscles is palsied; pupillary symp- ns may be present; the reflex processes and nutrition depend the downward progress of the lesion. If the bleeding occurs ve the origin of the phrenic nerves, a rapid death by asphyxia nevitable.

In a few cases (Monod, Oré, Breschet—quoted in Levier) the orrhage has been found restricted to one *lateral half* of the l, with the characteristic symptoms of Brown-Séquard's spi- hemiplegia (paralysis of the side corresponding to the lesion, sthesia of the other side).

ourse, duration, termination.—The course depends on the and location of the hemorrhage, and in part also on the ediate cause. In severe cases, especially of diffuse central ding, the fatal termination occurs very soon through paraly- f respiration; or secondary changes, acute gangrenous bed- s, pyæmia, and septicæmia soon cause death amid fearful ering. Charcot believes that a true hæmatomyelia is *always* l, but we cannot assent to this.

f the bleeding is small, the case may be protracted a very time, until at last death occurs from bed-sores, cystitis, r, marasmus, and other complications.

Partial recovery often occurs; the lesion in the cord becomes trized, and is restored to as normal a condition as is possible. ility and sensibility return, at least in part; the bed-sores l, the vesical palsy disappears, and the general health is good. But usually some muscles or groups of muscles remain paralyzed and atrophied.

Complete cure is doubtless rare, and only possible when the clot is very small. It is difficult to prove that it has taken place, although reports of cases and autopsies are very decidedly in favor of it.

The duration must vary greatly. Rapid cases terminate in a few minutes, hours, or days, while less severe ones require weeks, months, or even years before death appears, or a moderate degree of improvement is gained.

Diagnosis.

This is chiefly based on the *sudden and very rapid invasion of paraplegia* without much motor irritation, upon the *immediate severity of the symptoms*, and the *very severe and protracted course* of the disease. The decision may be supported by the existence of a reasonable cause, by certain prodromic symptoms, the absence of fever, and the elevation of temperature in the palsied parts.

The diagnosis may, at any rate, be difficult in slightly marked or complicated cases, and the disease may be confounded with various others which resemble it.

It is hardly possible to mistake it for *cerebral apoplexy*. The retention of consciousness, the absence of all symptoms of paralysis of cerebral nerves, the paraplegic form assumed by the disease, the paralysis of the sphincters, etc., must guard us from this. And even in difficult cases, such as certainly occur, we shall not fail to make a correct diagnosis if we carefully attend to the symptoms.

The diagnosis from *meningeal hemorrhage* will usually be easy (see above); in the latter there are active symptoms of irritation, hyperæsthesia and pain, violent spasmodic symptoms, while paralysis is less prominent; the disturbances of sensibility are quite slight, and the course of the attack is rapid and favorable. In hæmatomyelia the severity of the paralytic symptoms is very striking, while the phenomena of irritation are quite in e background; bed-sores appear quickly; the disease is severe, ten fatal, and often leaves incurable palsy behind.

The chief difficulty is that which attends the diagnosis from *ute central myelitis*, especially as the hemorrhagic form of the tter is included in spinal apoplexy. In both cases there is a estruction of the central gray substance, and the only point of istinction consists in the *rapidity with which the symptoms evelop.* The paraplegia requires hours or days for development n simple myelitis, in hæmatomyelia minutes or quarters of an our. The same is true, of course, in hemorrhagic myelitis. Ve may say that the greater the rapidity of development, the nore prominent is the element of hemorrhage. In distinguishing entral myelitis from spontaneous hæmatomyelia, we must atend to some other points; myelitis begins with symptoms of rritation, pain, slight spasm, the vertebræ are sensitive to presure, fever may be present, and anæsthesia and paræsthesia, parial palsy and weakness of the bladder precede the occurrence of evere paraplegia. The ascending spread of central myelitis may so be contrasted with the stationary nature of the symptoms in emorrhage. By the aid of these points we may distinguish ematomyelia from central myelitis, and in the latter we may parate the simple from the hemorrhagic form.

Poliomyelitis anterior acuta (or acute spinal paralysis) in lults is often quite like hæmatomyelia. It may, however, be fficiently distinguished by the usual presence of fever at the mmencement, the entire absence of all sensory disturbances, e absence of palsy of the bladder and of bed-sores.

The diagnosis from *ischæmic* paraplegia will usually be easy; though at the beginning it much resembles hemorrhage, yet vere ischæmic paraplegia occurs only in cases of obstruction of e aorta, and the latter may easily be recognized by the pathogomonic symptoms of absence of the femoral pulse, and disrbed circulation in the legs, etc.

The diagnosis of the level of the seat of hemorrhage is made y attending to principles already stated.

Prognosis.

Although we by no means agree with Charcot's view of the usually fatal character of hæmatomyelia, yet the prognosis is almost always very serious. Large central hemorrhages are always fatal. The same is true when the seat is at a very high level.

If the first few days and weeks pass without bringing very severe complications, the prognosis becomes gradually more hopeful. But complete recovery is rarely to be expected.

But even when the disease appears favorable, and the symptoms are slow in developing, a turn for the worse is always possible. Besides the formation of bed-sores, the chief thing to fear is the ascending spread of central myelitis.

Small circumscribed hemorrhages are certainly less dangerous —if they can be recognized at all during life.

For the rest, a prognostication cannot be made in any case without a careful weighing of all circumstances.

Treatment.

The prophylactic measures are inferrible from the etiology. Such causes as can be reached will be attacked. Retained or suppressed menses, a cessation of hemorrhoidal bleeding, heart-disease, congestion of the cord, etc., will also receive careful attention. It is especially needful to inquire whether the symptoms are related to a central myelitis, in which case all mean will be used, including large bleedings, cold applied to the back powerful derivation, mercury, iodide of potassium, etc.

For the hemorrhage itself, little can usually be done; by th time the physician arrives, it will (unless very considerable) us ally have ceased of itself. It will, however, be proper to tak measures for preventing a recurrence or an extension to oth parts of the cord, especially when signs of plethora, of excit action of the heart, of severe spinal congestion are present; w should then proceed with vigor to the employment of local an general bloodletting, free application of cold, maintenance of uiet position upon the side or abdomen, with the internal use f digitalis or ergot (or still better, subcutaneous injections of rgotin), purges, application of warmth to the extremities; these re the remedies here applicable, and from which the proper election must be made in each case.

Afterwards we have to treat the sequelæ. Secondary myelitis ust be checked by the use of such means as are indicated in he appropriate section. But the chief object will lie in the prevention of severe trophic disturbances, cystitis, bed-sores, etc., which are the most threatening complications. Nothing but the most *careful and self-sacrificing attention* can accomplish this; or directions see the General Division of this work (p. 192 et seq.)

If the first weeks pass without serious results, we may attempt to promote the resorption of the extravasation and the repair of secondary myelitis by *iodide of potassium*. For the same purpose we would advise the use of *lukewarm baths*, *warm springs*, and *brine baths*, or a moderate cold-water cure, and above all, the scientific application of *galvanism*.

The latter is also the chief remedy for the palsies, atrophies, and anæsthesias which remain even after comparatively favorable cases.

Special indications may of course arise in each case, which need not be enumerated here.

4. *Wounds, Crushing or Tearing of the Cord* (*Acute Traumatic Lesions*).

Ollivier, loc. cit. I. p. 246.—*J. Hahn*, Paraplégies par cause externe ou traumatique. Thèse. Strasb. 1866.—*Leyden*, loc. cit. I. pp. 310 and 321; II. pp. 84 and 139.—*M. Rosenthal*, l. c. p. 331.—*E. Gurlt*, Handbuch der Lehre von den Knochenbrüchen. II. 1. 1864.—*Lente*, Recovery from Fracture of the Spine. Amer. Journ. Med. Sci. 1857. Oct. p. 361.—*Ruehle*, Greifsw. med. Beitr. 1863. I. p. 12.—*Vogt*, Lähmung der vasomotorischen Unterleibsnerven nach Rückenmarksverletzung. Würzb. med. Zeitschr. VII. p. 248. 1866.—*Quincke*, Einige Fälle excessiv hoher Todestemperatur. Berl. klin. Wochenschr. 1869. No. 29.—*Fronmueller*, sen., Die Rückenmarkszerreissung. Memorabil. 1870. No. 12.—*M'Donnel*, Fracture of the Spine. Dublin Quart. Journ. 1871. Vol. 51. p. 215.—*W. Mueller*, Beitr. zur pathol. Anat. und Physiol. des R.-M. Leipzig,

1871. Beob. 1.—*Nieder*, Lowered Temperature in Injury of Spinal Cord. Med. Times. 1873. I. p. 154.—*Steudener*, Zur Casuistik der Herzwunden (Sch auch durchs R.-M.). Berl. klin. Wochenschr. 1874. No. 7.

We here include a large group of disturbances, in the full consciousness that the bond which unites them is somewhat loose. All, however, have one thing in common ; they possess an *acute traumatic lesion of the substance of the cord*, which leads to a destruction of the organ, usually limited as to its longitudinal extension, of variable extent in the transverse direction, and inevitably followed by a traumatic myelitis, similarly localized. This circumstance imparts to all these lesions (they include wounds by cutting, stabbing, and shooting, compression, crushing, and tearing of the cord) a great general resemblance, which from a practical point of view justifies their association in one group.

Etiology.

Severe traumatic lesions of the cord are hardly possible, unless the bony envelope, the spinal column, is injured at the same time.

There are but few spots (in the upper cervical and the lumbar regions) where the instrument of offence or the foreign body can reach the cord without injuring the spine, by passing in through the vertebral fissures.

It may be regarded as a very rare occurrence for severe traumatic lesions of the cord to take place without considerable injury to the bones or the soft parts.

By far the most common and important causes of these lesions of the cord consist of *fractures and luxations of th vertebræ.* All injuries which occasion these fractures or luxations may, therefore, be regarded as remote causes of lesion the cord.

Wherever fracture of a vertebra occurs, with displacement the fragments towards the vertebral canal, wherever in luxati the vertebræ are so transposed as to narrow the vertebral can there it is inevitable that severe lesions of the cord, compressi and crushing, or even local destruction and tearing apart, shou

r. Owing to the width of the spinal canal and the loose
hment of the cord, a considerable intrusion is necessary
re the latter is likely to be injured; and yet the lesions we
mentioned are among the most common sequelæ of frac-
s and dislocations of the vertebræ. Hence the great danger
ected with these surgical events.

e have no space here, nor are we called upon, to enter upon any details respect-
e causes and the occurrence of vertebral fractures and luxations, the displace-
which occur, their degree and direction, and their consequences. The reader
erred to the proper works on surgery, especially the exhaustive treatise by
. It is sufficiently evident that lesions of the cord may occur in cases of appa-
spontaneous injury to the vertebræ, such as the sudden breaking down of
s bone.

hat these traumatic lesions may occupy any conceivable sit-
n is plain; they have been observed from the atlas and axis,
racture and dislocation of which are usually so rapidly fatal,
n to the lumbar spine and even the sacrum, in any of which
ns the cord or the cauda equina may be more or less in-
ed.

n the second place, we would mention *gunshot injuries* of
cord, which form an important chapter in military surgery.
y are probably always complicated with gunshot fracture of
vertebræ, and the lesion of the cord is due either to the latter
irectly to the entrance of the ball into the medulla. These
s, therefore, always involve complicated fractures of the ver-
æ, with wounds which are often gangrenous, contain various
ign bodies, etc.; in short, all possible unfavorable circum-
ces.

ot every shot which strikes the spine is equally dangerous
e cord; the lesion is not rarely confined to meningeal hemor-
e, concussion, etc., which are infinitely less dangerous than
l wounds of the cord.

tabs and *cuts* of the spinal cord are rarer. Blows from
s, swords, and daggers have been repeatedly observed to
the cord, the point of the instrument having entered the
either by dividing the vertebral arches or by passing
gh the intervertebral spaces. The injury may vary in
t; the point may have merely entered, or the cord may be

cut through in various ways, partially or wholly. The foreign body (point of a sword, broken knife-blade) may then remain sticking in the cord or vertebræ. In like manner, in fractures, pointed fragments of bone may injure the cord and maintain a permanent irritation.

Finally, *severe concussion of the body*, such as is produced by a heavy fall on the back, rump, or feet, or by the striking of heavy bodies upon the back, has been observed in a few cases to be followed by considerable lesions of the cord (extravasation, destruction, etc.) without injury of the vertebral column. Thus, Fronmueller found a complete mashing of the dorsal medulla of the extent of three and one-half centimetres in a person upon whose back a heavy beam had fallen without injuring the spinal column. These cases may also be counted in with the severest forms of spinal concussion, and can only be distinguished from the latter by the coarse anatomical lesion. Parrot once found the cord of a new-born infant torn apart by excessive traction during labor.

Pathological Anatomy.

The lesions of the spine and other neighboring parts which cause or accompany injury to the spinal cord need not occupy us further; we refer our readers to the text-books on surgery and pathological anatomy.

In the cord itself we observe various phenomena :

1. *Simple incised or punctured wounds.* At first a wound o various size and depth, filled and closed with curdled blood the edges of the cut often projecting over the pia. A foreig body (point of a knife or dagger, splinter of bone) is often foun in the wound. The size of the wound differs: it involves variou portions of the transverse section of the cord, one or the oth white column, more or less of the gray substance; often o lateral half is severed, rarely the whole (J. L. Petit, Vogt).

In a few days, and at subsequent periods, the edges of th wound are still further protruded, are colored a brown-red, an more or less covered with pus; their vicinity is hyperæmic, mor or less softened; filled with little extravasations of blood; puru

tion or actual abscess of the cord is rarer. The menin-
same time, are reddened and inflamed, covered with
ent exudation, and abound in capillary hemorrhages;
ce they are opaque, thickened, adherent; the spinal
reased, turbid, reddish.

often been demonstrated in animals, and is probable,
sufficiently proved, in the case of man, that in favor-
ces the edges of the wound heal, and a cicatrix of
tissue repairs the injury. It is not yet certain how
e-elements are restored.

hing of the cord produces a softening and disintegra-
ponding to the size of the crushing body, usually
with hemorrhage, but not always with tearing of the
The medullary substance is changed into a soft black-
colate-colored mass, sometimes rather grayish, com-
ood and débris of nerve-substance, showing its color
e pia, which is suffused with blood to a greater or
. The crushed spot is usually flat, constricted, and

yperæmia presently appears in the parts adjoining,
essive inflammatory softening; the medulla swells,
s seen in section become obliterated, the consistency
lulla diminishes; it imbibes at first a reddish, after-
llowish tint, and is full of small extravasations. The
shows in the immediate vicinity many granular-cor-
tritus of myelin, decomposed blood-corpuscles, pig-
blood-crystals; also inflammatory swelling, and after-
k-down of nerve-fibres, axis-cylinders, and remnants
n-cells; in short, the ruins of the medullary substance
ith the products of its own acute traumatic inflam-

few weeks, a thin, gray-yellow, semi-fluid mass is
he place which was crushed, partially enclosed in a
of young connective tissue; the swelling of neigh-
ts continues, they are closely adherent to the mem-
ir color is grown paler, and is now a grayish yellow;
demonstrable simple softening is prolonged to some
bove and below. The lower portion of the cord is

especially apt to be found softened throughout its whole extent. Actual abscesses are seldom found. The characteristic secondary degeneration of the posterior and lateral columns, ascending in the former and descending in the latter, is a pretty regular occurrence.

If the wounded man lives longer, the destroyed medulla is by degrees absorbed, and in its place a sort of cicatrix is formed of young connective tissue, abounding in fluid, which grows harder and encloses here and there cystic spaces of various sizes. A full regeneration of the nerve-substance is not known to occur in man.

3. *Complete severance of the cord* is recognized by the fact that the two ends of the cord are separated by a space which may amount to three centimetres or more, in which case the pia is, of course, also torn, while the dura may remain uninjured, or nearly so.

The space is filled with a semi-fluid mass, partly composed of blood, at first dark, afterwards rather of a chocolate or gray color. Inflammatory softening occurs, as after crushing, and extends more or less upward and downward. If the patient lives long enough, the commencement of cicatrization and junction of the ends may be observed.

4. *Hemorrhagic destruction* of the substance, caused simple concussion, is quite like a hemorrhagic softening with its consequences.

The secondary changes in the other organs of the body, b sores, cystitis, disease of the kidneys, etc., are the same as other forms of severe spinal paralysis, and will be more fu described under myelitis.

Symptoms.

For the sake of distinctness in the presentation, we make groups, the one (*a*) containing *comparatively slight injurie the cord, the simple incised and punctured wounds;* the ot (*b*) embracing all the *more serious lesions, squeezing, crush and tearing of the cord.* It need hardly be said that betw these two classes there exist transitions, and numerous analog both as respects the anatomical changes and the symptoms, course, and termination of individual cases.

a. The symptoms which indicate that an *injury by cutting, or stabbing, in the neighborhood of the spine, has penetrated the cord*, will be at first those of partial or total interruption of conduction in the cord, which extends to the portions behind the seat of injury, and is not merely confined to the district of the nerve-roots which lie at the point of injury.

This interruption of conduction may vary in extent and distribution, according to the seat and extent of the injury. Almost all the experiments in the physiology of the cord consist in the infliction of such simple injuries by section, and all varieties of symptoms may be imagined in connection with them.

At the moment the injury is received, there is usually a motor paralysis of various extent in the form of paraplegia, or spinal hemiplegia, or hemiparaplegia, or even paralysis of the trunk, and all four extremities. With this is usually associated *sensory paralysis*, due to the injury, and correspondingly of very various extent. It may be paraplegic, or may be limited to one side, and in the latter case is upon that opposite to the lesion and the motor palsy; it may be quite circumscript; it may affect certain qualities of sensation only, as the sense of touch, the muscular sense, etc. In cases of very restricted lesion, *hyperæsthesia* (in the form of a girdle, or otherwise distributed) is often found.

If the injury is of any considerable extent, *paralysis of the ladder and rectum* is always present; at first with complete etention of urine, which soon gives place to equally complete ncontinence, while the evacuation of fæces takes place involun- rily and unperceived. Close examination usually detects also *aso-motor palsy* (elevation of the temperature of the skin, in- reased redness) in the regions affected by the motor palsy.

The *reflex actions* are usually totally suspended at the first oment under the shock of the injury, but soon return, and ay become exaggerated. This depends, of course, upon the eat of lesion.

If we add to this the *girdle-pains* which are usually present, orresponding with the seat of lesion, and due to injury of the roots, and the symptoms which depend on injury of the bones and soft parts, we have a pretty complete picture of the condition of the patient during the first few days after the accident.

This, however, is soon complicated by the symptoms of secondary *traumatic myelitis.* Usually the inflammation is transverse in its distribution, covering nearly the whole breadth of the cord, but having little extension in the longitudinal direction. The fever usually lasts but a few days. Striking symptoms of *irritation* now commonly appear; pains encircling the trunk like a girdle, active pains in the paralyzed parts, hyperæsthesia of various extent in the skin, also spasmodic conditions, twitching and contractures of single muscles and groups. The palsy at the same time increases rapidly in the transverse direction without extending much upwards, *i. e.*, it seizes by degrees upon the motor paths passing the injured spot, which were at first unaffected (the sensory and vaso-motor paths, bladder, rectum, etc.), without any considerable change in the upper limit of the lesion. The reflex actions are decidedly increased, but may at a later period cease entirely, when the process has extended to the bot tom of the cord. If foreign bodies remain in the wound, th symptoms of irritation reach a still higher point, with ver severe pains, violent spasms and contractures.

At a later period bed-sores, with all their consequences, a pear—pyæmia and septicæmia, cystitis, etc.

According to the height at which the lesion is situated, oth symptoms may arise to complicate the case; we need not me tion them all. The higher the injury, the more prominent a the disturbances of respiration, and the more threatening th import.

b. The symptoms which, accompanying severe lesions of t spine or other places, indicate that the cord is *crushed or to* are usually those of complete and very severe paraplegia. T posterior portion of the body is absolutely paralyzed, with c responding complete anæsthesia, limited quite sharply abo the reflex actions are depressed or suspended, seldom increase the bladder is paralyzed to a severe degree, causing retention urine and frequently an enormous distention of the organ; t intestine is paralyzed, producing meteorism; the rectum is pa

e symptoms follow naturally from the lesion of the cord. The motor,
d vaso-motor palsies are direct consequences of the interruption of con-
the cord. The suppression of reflex acts which is found even where
entres are not directly injured, is the result of the severe shock suffered
l; after the lapse of some hours or days the reflex centres recover, the
ns reappear, and may even exhibit considerable increase if circumstances
he same is true of the vaso-motor centres, especially for those which
r the evacuation of urine, situated in the lumbar cord; the shock, com-
the crushing, paralyzes these centres first, whence the complete retention
the excessively distended bladder, which exists even in cases where the
d is not the seat of the crushing lesion. If the centres recover, occa-
plete evacuations of the bladder occur quite involuntarily, and usually
l; thus, Steudener found a powerful contraction of the bladder occur-
rly as soon as the catheter in the urethra irritated the fossa navicularis—
on which is in perfect accordance with physiological facts observed by
a later time the urine begins to drip continually.

pism which is so frequent an occurrence in severe fractures of the verte-
nore difficult to explain. It is most common in connection with crushing
ical portion, rarer with that of the dorsal region, and never occurs with
om the third lumbar vertebra downwards. In many cases the erection
served directly after the injury; ejaculation at the same time has oc-
few cases. The erection is either powerful, or lax and incomplete; it
t felt at all, or is painful; at a later period it may diminish, and may
oduced by catheterism or other irritations.

ot hard to form a plausible theory of the occurrence of this phenomenon,
nown facts; though it must be confessed that the subject is obscure in
ects. It is best explained by the assumption of an irritation at the

ignorant of the precise condition of the latter centres and their reflex excitability in cases of permanent erection. If we take into view the symptom of ejaculation, which can certainly be nothing but a symptom of irritation, the assumption of irritation of the paths which give rise to erection will be found the more plausible. We cannot see how in such cases priapism can be regarded as a phenomenon of paralysis, unless it be of vaso-motor paralysis. At a later period—and perhaps at the beginning in many cases—there is no doubt that reflex irritation (as that originating in a distended bladder, the introduction of a catheter, bed-sores, etc.) may give rise to, and sustain, the erections.

If the above group of symptoms should allow the slightest doubt of the existence of a severe lesion of the cord, if the case should at first be taken for one of severe simple concussion, a few days' delay will usually bring full evidence of the real condition. At that time fever appears; the symptoms of acute traumatic myelitis become more and more distinct; bed-sores appear, and increase rapidly and steadily; if the lumbar region is crushed, there is rapid atrophy of the leg-muscles with loss of electrical reaction; the urine becomes bloody, purulent, ammoniacal, etc. The temperature, especially when the cervical region is crushed, often rises continuously and to an excessive height (43°–44° C. [109° to 111° Fahr.], Brodie, M'Donnel, Quincke, and others)—the rise which occurs in the neuro-paralytic agony. In other cases, when the dorsal portion has been injured, the temperature has been abnormally low for some days before death (Nieder). For the interpretation of these symptoms we refer to page 128 et seq.

Thus the most severe cases are rapidly brought to an end by complications—such as palsy of respiration, disturbances of circulation, pyæmia, neuro-paralytic agony, and so forth.

The disease is by no means, however, always so severe; there are cases of partial crushing, where a fortunate accident has saved a portion of the transverse section from destruction, of which the result is partial paralysis (Ollivier, Obs. 25 and 26). The entire process is milder and less dangerous. This is not the place even to touch upon all the possible cases of such partial lesion; we need only say that the literature of the subject of fractures of the vertebræ contains cases of all possible degrees of severity.

The symptoms differ, of course, in correspondence with the level at which the lesion may be seated.

cervical portion is affected, death usually occurs at he lesion is at the height of the first or second cervical *e. g.*, in luxation of the odontoid process; and death as speedy when the lesion is above the origin of the ierves. All four extremities are then paralyzed, while ration is so difficult as only to be maintained by the d anxious action of the auxiliary muscles of respira- speech and voice are weak, and swallowing is difficult. vical enlargement below the phrenic nerves is the seat the expiration is chiefly affected; the legs are wholly l, the arms more or less completely so; the sensibility ns may be partially retained; reflex actions are retained, ggerated; M'Donnel even saw co-ordinated reflex actions hand, in a state of absolute palsy, made a movement to genitals during the act of catheterization); priapism is uent; life may be retained for a considerable time.

lesions are in the *dorsal* region, the arms are free, and is palsied, up to an uncertain height; the legs are more aralyzed, often unilaterally, with crossed anæsthesia; ation is less impeded, the voice and speech are some- cted; the reflex actions are retained and exaggerated in quent course of the disease; the bladder and rectum yzed; priapism is somewhat rare; bed-sores follow.

ion of the *lumbar region* the arms and a great part of are free. The legs, bladder, and rectum are totally l; respiration is not impeded; reflex actions of all sorts y extinct; no erections; rapid atrophy of the muscles, iction of their electrical reaction. The symptoms of the *cauda equina* are quite similar; but the freedom plication of certain nerve-paths, especially those belong- e lumbar plexus, often enables us to refer the seat of the cauda equina.[1]

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[1] , Ueber acute Spinallähmung bei Erwachsenen. Arch. f. Psych. u. Ner- V. p. 785. Beob. VI.

Course, Duration, Termination.

There are hundreds of physiological observations which show that improvement, and even cure, may occur in the first and slighter form of lesion (simple incised wounds of the cord). Goltz and Freusberg have kept dogs alive for many months, even when the dorsal cord had been completely severed by a cut; they observed, however, no occurrence of regeneration.

An absolutely fatal termination, therefore, even in man, can hardly be predicted; indeed, there are numbers of cases of undoubted lesion of the cord, in which a comparative cure was effected and life was retained for many years. Such are the cases of cure of a probable punctured wound, given by Ollivier, and of various incised wounds, by Brown-Sequard and others. These, however, are the exceptions, which must not be looked for unless in slight injuries. If such is to be the result, the symptoms of myelitis do not become severe, they soon pass off, and the outer wound closes; the symptoms of palsy are relieved in part, and in part remain, so that a more or less complete cur is by degrees obtained. The importance of the functional repar tion of the lesion, urged by Schiff (p. 64 above), can hardly overlooked.

Usually, however, the secondary myelitis continues to increa the paralysis increases, bed-sores, with their unfortunate cons quences, appear, and the fatal end is reached after more or le protracted sufferings.

The second and more severe form is almost always fatal. Wh the cord has been thoroughly crushed or torn in any part it hardly possible to conceive of regeneration; and even if life be tained, in a number of cases, for weeks or months (M'Donnel, t months; Steudener, fifteen weeks; Page, in a case of severance the cervical cord between the fifth and sixth vertebræ, even f teen months), yet the ultimate result is pretty certain, and occu with the usual symptoms of severe spinal palsy. It is a matt of course that death may occur in a few hours or days after t injury by paralysis of respiration, shock, or similar occurrence

Is a cure ever effected? and is it possible in any severe spin on? The opinions of physiologists are divided upon this point above, pp. 64, 65). There are no convincing proofs in the of man. In the instance given by M'Donnel, a sort of cica- was found at the close of two months, but not a trace of ve-tissue could be found in it. Ollivier's observation (No. 18), Lente's case, are strongly in favor of the repair of lesions of lerate severity.

Diagnosis.

It is not difficult to recognize an injury of the cord by the ceding symptoms.

In case of simple injury to the meninges, by puncture or in- on, the occurrence of *meningeal apoplexy* might give rise to mistaken diagnosis of an injury of the cord. The former will ly be recognized by the symptoms of irritation, which are minent at the outset—pain, spasmodic phenomena—and by slighter degree and the greater diffusion of the paralysis; finally by its rapid and favorable course.

n severe injuries of the cord, the existence of *spinal apoplexy* be suspected; but it is of no importance to determine it, as a ing of the cord is doubtless always attended by hæmato- ia, and the symptoms and course of the two diseases are tially similar.

ases of *severe concussion* of the cord may usually be re- zed by the absence of a clear demarcation of the anæs- a and the palsy, by the subsequent course, the absence of ores, etc. If dislocations of the vertebræ can be proved to the crushing of the cord will become more probable.

Prognosis.

his will readily be inferred from what has been said. It is dubious, even in the slightest cases of injury, and we should epared for a fatal result through secondary myelitis; but is still some hope.

all severe cases of crushing and tearing of the cord, death nost absolutely certain, sooner or later. But there are exceptions, as has been before said, in the case of slight and very partial lesions only.

Treatment.

The first thing to be attended to is a careful treatment of the *external injuries* (wounds, fractures, and luxations of the vertebræ), for the details of which we refer to the hand-books of surgery.

The injury to the cord introduces a very important element of danger in all these cases; in most instances it is the cause of death; we ought, therefore, to make every effort to relieve it or to promote functional recovery. In addition to the ordinary surgical methods, we shall often have to consider the propriety of *trepanning* the spinal column for the relief of compression of the cord, the removal of fragments of bone, replacement of the ends of the fractured bone, etc. We need not, however, discuss the value or the feasibility of trepanning, or its indications, for which we can refer to the work of Gurlt. And yet we believe the operation is indicated wherever there is good reason to hope for gaining anything in the treatment of the dislocation which caused the lesion of the cord; and such gain would be more probable in fractures of the arches of the vertebræ, as fragments of the bodies can hardly be reached. We can scarcely ever expect any direct result, as the injury to the cord is not from simple compression, but usually from crushing or tearing. Yet the removal of displaced fragments will at least increase to some extent the chances of life. We should not, therefore, hesitate, in so almost hopeless a case, to undertake an operation not of itself very dangerous, if there is any possibility of relief. The physician must carefully weigh all the chances; it is certain that in all severe cases the operation will do little harm, and perhaps some good.

In the second place, we have to consider remedies for *traumatic myelitis*, local (or if necessary general) bleeding, cold, ergotin, and belladonna, frictions with mercurial ointment, etc. (See the Treatment of Acute Myelitis.)

By far the hardest task will consist in the personal attentions to the patient, those, namely, which are required for the prevenon of bed-sores and cystitis, which usually destroy life. Two ontradictory requirements are usually present: absolute quiet, or the surgical injury, and frequent change of posture, for the revention of bed-sores. We must try to do what we can with ater-pillows, air-cushions, pillows pushed in alternately on oposite sides, cushions stuffed with chaff, great cleanliness, position on the face, etc., as circumstances dictate.

The treatment of the vesical symptoms, the fever, marasmus, pains, etc., depends on general principles. We should compare the treatment of acute myelitis.

5. *Slow Compression of the Cord (Chronic Traumatic Lesion of the Cord).*

Compare the works of *Ollivier* (I. p. 387), *Hasse* (p. 735), *Jaccoud* (Des paraplégies, etc.), *Brown-Séquard* (Paralysis of the Lower Extremities, etc. 1861), *M. Rosenthal* (p. 313), *Leyden* (I. pp. 213–311; II. p. 147). Also

Charcot, De la compress. lente de la moëlle ép. Leçons sur la mal. du syst. nerv. II. Sér. II. fasc. 1873.—*Bouchard*, Compress. lente de la moëlle. Dictionn. encyclop. des sc. médic. II. Sér. Tom. VIII. p. 664. 1874.—*C. Hawkins*, Cases of Cancerous, etc., Disease of the Spinal Column. Med. Chir. Transact. XXIV. p. 45. 1841.—*Vogel* und *Dittmar*, Deutsche Klinik. 1851. Nr. 38.—*Traube*, 5 Fälle von Rückenmarkskrankheiten. Charité-Annalen. IX. 2. p. 129. 1861.—*Ruehle*, Zur Compression des R.-M. Greifsw. med. Beitr. I. p. 5. 1863.—*James Young*, Case of Temporary Paralysis. Edinb. Med. Journ. May, 1856.—*Ogle*, Case of Paraplegia, etc. Transact. Path. Soc. XIX. p. 16. 1868.—*A. Joffroy*, Cas de fract. de la colonne vert. Arch. de Phys. I. p. 735. 1868.—*Loudet*, Curabilité des accid. paralyt. conséc. au mal vert. Mém. de la Soc. de Biol. 1862–1863.—*Michaud*, Sur la méning. et la myélite dans le mal vertébr. Paris, 1871.—*Charcot*, Anat. pathol. et trait. de la parapl. liée au mal de Pott. Gaz. méd. 1874. No. 49.—*A. Courjon*, Parapleg. dans le mal de Pott. Paris, 1875.—*E. Rollett*, Wien. med. Wochenschr. 1864. Nr. 24–26.—*De Giovanni*, Storia di un caso di paraplegia, etc. Riv. clin. di Bologna, 1870. No. 12.—*Leyden*, Ueber Wirbelkrebs. Charité-Annalen. XI. 3. p. 54. 1863.—*M. Rosenthal*, Wiener med. Presse. 1865. Nr. 42–45; Zeitschr. f. prakt. Heilk. 1866. Nr. 46–51. *Tripier*, Du cancer de la colonne vertébr. Paris, 1866.—*Th. Simon*, Paraplegia dolorosa. Berl. klin. Wochenschr. 1870. Nr. 35 und 36.

Definition.—Numerous as are the conditions which we embrace under this title, yet all have this in common, that *a power cting from without upon the cord (and the nerve-roots) com-*

presses it very slowly and gradually in a limited longitudinal extent, giving rise to a series of characteristic symptoms which in all cases may be recognized by their essential features, and often are the first to call our attention to the commencement of a severe affection.

These symptoms, however, are not usually produced by the pressure as such, or exclusively, but by the *subacute or chronic transverse myelitis which develops almost invariably at the point of compression* and by the *secondary degeneration* of the cord, extending upwards and downwards.

The essential lesion is, therefore, a circumscribed transverse myelitis, the so-called *myelitis from compression;* and we should not devote a separate section to it, were it not that it forms a connecting link between very various morbid states originating in the spinal column and its contents, and that the symptoms of myelitis from pressure compose a very important feature, common to all those forms of disease.

Etiology and Pathogenesis.

Any circumstance which very gradually narrows the spinal canal, usually in the way of organic growth, and thereby leads to a slowly increasing local compression of the cord, may become a cause of myelitis by compression.

We have already mentioned an important part of these causes, viz., *meningeal tumors*. To avoid repetitions, we would refer to what has been said above (see page 262 et seq.) upon this point. We here repeat that a gradual compression of the cord may be occasioned not solely by the so-called neoplastic tumors proceeding from the spinal membranes, but equally well by meningeal tumors formed by inflammatory and hemorrhagic processes, parasites, and the like, and by tumors which originate in the perimeningeal tissue. All these tumors have one thing in common, that their expansion is very soon checked by the narrowness of the canal, and that they then begin to exercise an increasing pressure upon the cord. It is self-evident that this pressure may affect the cord in various ways and from different d may thus produce a great variety of symptoms, but tial conditions remain the same.

y be questioned whether the *intra-medullary tumors*, veloping within the substance of the cord, ought to be among the causes of compression. They do not act hout, and therefore differ somewhat in their symptoms. racteristic of them that the cord is gradually compressed point of its transverse section, with myelitis as a genempaniment; for which reason the characteristic initial ns relating to the compressed roots are absent, and those ression of the cord begin at once. For all details we he section on tumors of the cord, and premise only this that all sorts of intra-medullary tumors may produce ptoms of myelitis by pressure—as glioma, sarcoma, , tubercle, syphilitic gummata, cysts (hydromyelus and yelia), etc.

r the most important cause is that furnished by *diseases rtebral column.* The most frequent of these, and also t frequent cause, on the whole, of myelitis from com- is *caries of the spinal column* (Pott's Disease, Spondyce). We need not describe this particularly, for it is ated of in the text-books of surgery; our present object ed to inquiry as to the manner in which it may give rise compression of the cord.

e first place, the wasting and sinking of the bodies of bræ produce *kyphosis*, which may give rise to a *bend- narrowing of the vertebral canal*, with compression of . This is certainly very rare, and only occurs in exces- hosis. Enormous angular kyphosis may occur without ptoms of compression of the cord; and on the other e paraplegia accompanying kyphosis often disappears er without the least change occurring in the latter; and here are cases of vertebral caries without any kyphosis, e nevertheless accompanied by paraplegia. The kypho- , therefore, the sole nor even the chief cause of compres- he cord.

ost cases this compression is produced by the *inflamma- dation which accompanies the caries.* The masses of pus produced by caries, and in particular the fungous granulations, accumulate between the dura and the bodies of the vertebræ and press the dura inwards; they produce a pachymeningitic growth and thickening of the dura, causing a contraction of space in the vertebral canal (Charcot, Michaud; see also the very artistic illustration in Ogle). The dura is thickened; its outer layers especially are changed to a proliferous mass of young fibro-plastic tissue, partly undergoing caseous metamorphosis, which either surrounds the dura at one point like a ring, or presses it from one side against the vertebral canal to the extent of ten, fifteen, twenty millimetres or more. The nerve-roots at the place are more or less implicated, thickened, swollen, inflamed, etc. Simple caseous pus-deposits may become a cause of pressure, or the protruding intervertebral cartilage, or separated and displaced fragments of bone.

In the second place, *carcinoma of the vertebræ* should be mentioned as a frequent cause of compression, chiefly observed in old persons. The primary as well as the secondary disease may act in this way; the latter quite usually follows primary cancer of the breast, but also that of any other organ. Not all cancers of the vertebræ produce spinal symptoms; that depends of course, upon their location, size, and the direction taken i growing; but when cancer attacks the arches of the vertebr and the nerve-roots which pass between them, when it has a tacked, softened, and thoroughly rotted the entire bodies of ve tebræ, when it presses in to the vertebral canal, seizes on t spinal membranes and grows directly against the cord itself, th the characteristic spinal symptoms usually appear, and the for of compression known as paraplegia dolorosa, with most acu pains, is developed.

There are several diseases of the vertebræ, much inferior in i portance to the preceding, which may occasionally lead to co pression of the cord. Such are *exostoses* of the vertebræ growi into the canal; *osteoma; syphilitic* new formations of the bon also *dry arthritis* of the vertebræ, when it leads to considerab swelling of the articular processes, osteophytic growths, depo tion of bone, etc. Here must be mentioned also the thickeni he odontoid process of the axis, anchylosis of the vertebræ, all similar affections.

inally, it remains to be said that *external tumors* of all , growing towards the vertebral column, and entering the bral canal by any natural or pathological opening, may rise to pressure of the cord. Such are echinococci, sarco- , aneurisms, etc.

ll these causes (with the partial exception of intra-medul- tumors) have one element in common, namely, that they on very gradually against the cord, reaching *first the nerve- and meninges*, irritating and afterwards compressing them, thus giving rise to a group of most characteristic symptoms, ch belong to the first period of development.

The cord itself is then attacked; it is exposed to a *gradual pression* which interferes with and interrupts the conduction. r is this all, for after the compression has lasted a longer or rter time (usually a very short time—sometimes even before aplegia exists—Charcot, Michaud) *inflammatory changes* ear; myelitis is developed, at first confined to the seat of pression, but usually extending over the whole transverse ion of the cord. With the compression and its concomitant litis, very characteristic symptoms are associated, belonging second stage of the disease. In most cases these symptoms to be connected with the appearance of myelitis.

he question whether compression may exist alone without itis, and may of itself produce the paraplegic symptoms, or her pressure-myelitis is a necessary condition, is somewhat perfluous one. It is certain that compression—especially be acute—is capable by itself of giving rise to the seve- paraplegia, which, if the compression be speedily and suc- ully removed, may disappear in a comparatively short time, the oft-quoted cases of Ehrling and Brown-Séquard. It is, ver, equally certain that in the very great majority of cases protracted compression of the cord is very soon compli- by pressure-myelitis; it does not seem probable to us that myelitis is the result of an ischæmia produced by compres- an ischæmic softening, but we consider it as the direct equence of the irritation of compression acting upon the elements of the tissue. Finally, it seems equally certain that in many cases the mere irritation of tumors pressing against the spinal membranes (carcinoma, etc.) is enough to produce myelitis without actual compression. We therefore see that the pathogenesis of the symptoms commonly known as compression of the cord may be of several sorts; they may arise through compression alone, or through compression and the consequent myelitis, or finally through myelitis alone.

In conclusion, if the compression is not soon relieved, and if myelitis is once established, *secondary degeneration and sclerosis* of the cord supervene, as in the case of every transverse myelitis (see farther on, No. 19), invading in a strictly regular way the various divisions of the cord above and below the point of compression. These in their turn lead to other definite symptoms, which may be considered as forming a third period of the disease.

Pathological Anatomy.

The pathologico-anatomical changes which belong to the primary lesion (vertebral disease, tumors, etc.) cannot be described here for want of space; the reader is referred to the text-books of pathological anatomy and surgery. We have mentioned a few chief points under the etiology, and it remains here only to describe what is directly concerned with the cord and its appendages.

The *meninges* are often hyperæmic, opaque, thickened, adherent to the neighboring parts; often covered with deposits of various thickness, which have much to do with the production of compression; often, however, they are remarkably little changed, are smooth, and simply pushed a little from their usual position.

The nerve-roots are almost always more or less involved in the morbid processes. They may be closely united with the tumor or the exudation, or may appear one with it; at first they are usually swollen, hyperæmic, inflamed, their fibres are in a state of fatty degeneration and beginning to break down, and this may be traced into the cord (neuritis); at later periods the roots are atrophied, pale, gray, degenerated, and nearly reduced to a connective tissue abounding in nuclei.

In carcinoma of the vertebræ, especially, the nerves and nerve-roots are reddened, swollen, fuller of fluid; rarely atrophied, and still more rarely directly involved in the cancerous growth. Simple contact with the malignant tumor is sufficient to produce severe neuritis.

The *substance of the spinal cord* is more or less flattened and thinned at the point compressed, is often reduced to a slender cylinder, hardly the size of a quill, or to a flat, ribbon-shaped string. The compression is sometimes greater from the front, sometimes from behind, sometimes from one side, and thus the cord gets a distorted, irregular look. The place compressed varies extremely in length; above and below it the cord is sometimes of its usual thickness, and sometimes rather thickened in a club-shape. The consistence of the compressed spot is usually lessened at first (inflammatory softening), but afterwards may be increased (sclerosis). The place is anæmic, pale, often presenting no distinct change to the naked eye; usually, however, the outlines seen in cross-section are quite indistinguishable, the medulla is cloudy, or, at a later stage is gray and translucent.

Microscopical examination shows—if the compression has lasted some time—a considerable increase and thickening of interstitial connective tissue at the place, and mingled with this are many granular corpuscles, often also corpora amylacea, while the walls of the vessels are thickened and in a condition of fatty degeneration; the axis-cylinders are often swollen, while some are broken down or have disappeared; in the ganglion-cells of the gray substance there may be observed swelling, the formation of vacuoles, pigment-deposit, and often a breaking down and disappearance (Michaud, Joffroy). In a word, there is chronic transverse myelitis, chiefly interstitial, extending over the greatest part or the whole of the transverse section.

The myelitic changes extend beyond the place crushed, and may be observed above and below at various distances, with diminishing intensity.

At a short distance from the spot, these changes usually appear confined to clearly defined portions of the transverse section, and in these may be followed up and down almost the whole length of the cord. These are the well-known *secondary degenerations* (Tuerck, see farther on, No. 19). Above the compression, the posterior columns are affected, higher up the fasciculi graciles alone, and in the latter the disease often ascends as high as the medulla oblongata. In the lower segment the lateral columns alone are affected, and chiefly their posterior part; the disease descends in this to the conus terminalis. The change may often be recognized in fresh preparations with the naked eye by the translucent, grayish, or slightly yellowish tinge of the tissue, but more usually the altered tissue cannot be recognized with certainty until the cord has lain some time in chromic acid, when it acquires a lighter color. With the microscope, interstitial proliferation of connective tissue, and degeneration of nerve-fibres are to be seen. In a few cases the degeneration has been seen ascending in the lateral columns also, usually but for a short distance (Michaud). Commonly the degeneration is not uniform in the two sides. The illustration (Fig. 5) gives a good view of the typical distribution of these alterations.

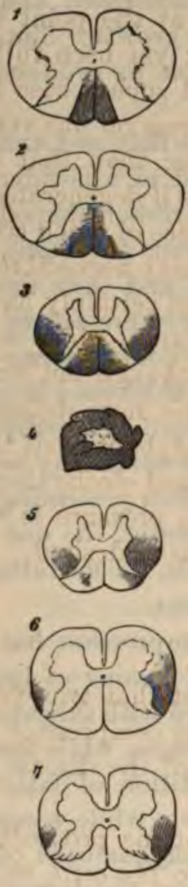

FIG. 5.
Compression of the Dorsal Medulla.
Diffuse myelitis at the point of compression (4); the right half is more compressed than the left. Above, secondary ascending degeneration in the fasciculi graciles (1—3): below, descending degeneration of the lateral columns to the lumbar cord (5—7). After Michaud.

The myelitic process often extends to great distances in the gray substance also, especially downward, but in this situation requires close microscopic examination to detect it; the characteristic signs consist of sclerosis of connective tissue, thickening of the arterial walls, atrophy and disappearance of the nerve-elements, pigmentation, etc. This process is important in explaining many later symptoms.

In severe cases all these processes make progress; there are consecutive changes in remote parts of the body (muscular atrophy, degeneration of peripheral nerves, cystitis, bed-sores, etc.), which hasten the fatal result.

In more favorable cases it is possible for restoration and full recovery to occur; this is certainly true in vertebral caries, and would doubtless apply to other cases if the cause of compression were removed. In such an event there must be a regeneration and restoration of nerve-elements, at least in part, at the point of compression. The processes, however, have not yet been fully studied. Charcot and Michaud have examined such a case in which recovery had occurred; the seat of compression was very distinct, its transverse section was much smaller than that of other portions of the cord, and it looked gray, as if degenerated. The microscopical examination showed the presence of much connective tissue, among which there were a great many nerve-fibres, normal in appearance, though slender; they must certainly have been diminished in number. In the gray substance, which was much reduced in size, there were found some, though not very numerous, ganglion-cells. Little is known of the more delicate changes in these processes of restoration; it is probable that the axis-cylinders of some nerves are not destroyed, and clothe themselves with a fresh medullary sheath when the pressure is removed. Further examination of the subject is to be desired.

Symptoms.

It is important to remember from the outset that two chief groups of symptoms are to be distinguished in compression of the cord, upon which may be based a division of the disease in two stages.

In the first group are included all the symptoms proceeding from *lesion of the parts situated externally to the cord*, especially the nerve-roots, meninges, bones, etc. They may also be designated as prodromal symptoms (symptômes extrinsèques, Charcot). This group furnishes the symptoms which are usually decisive of the differential diagnosis of the various causes of compression, while those of the second group are nearly the same in all cases, with the exception of such variations as depend on the level at which they occur in the cord, or the portion of the transverse section which is occupied.

This second group embraces all the symptoms which are derivable from *compression of the cord itself, and from the myelitis* caused by pressure (symptômes intrinsèques, Charcot). They are nearly alike under all possible causes, as above stated.

The succession and development of the symptoms of these two groups will give the characteristic signs of the disease.

This being premised, the *general symptoms* are as follows. After those of the primary disease (as Pott's Disease, vertebral cancer, etc.) have existed for some time, or even before they have been observed, the first evidence that the contents of the vertebral canal are attacked consists in certain *symptoms of irritation of all the organs contained* in the canal, the periosteum, meninges, and *especially the roots of the spinal nerves. Pain* of various sorts and degrees of violence, pains in the form of a girdle, excentric neuralgias in almost any spot (but holding constantly to that spot), open the scene; a great *hyperæsthesia* of those portions of skin which correspond to the distribution of the pain may be added, but often disappears to make room for a corresponding *anæsthesia*, often limited to a few islands of skin—or both conditions may subsist together. Severe *pain in the back*, local stiffness of the spine, great sensitiveness of the spinous processes, are seldom absent. The neuralgic pains are often accompanied by herpetic or bullous *eruptions of the skin.* To these are afterwards added (especially when the lesion occupies the cervical or lumbar enlargement) *states of motor irritation* in the districts of the nerves whose roots are first implicated, as twitching, spasm, tonic spasm, and contracture; *weakness and paralysis* is soon added, confined to certain muscles or groups of muscles, to one extremity or the other, and often accompanied by great *atrophy and loss of electrical excitability.* It must be observed that these sensory and motor symptoms are located in the same, or adjacent nerve-districts, and therefore may be referred to a single source of disease.

If the original causal lesion then declares itself by such signs as progressive kyphosis, congestive abscesses, pain strictly localized in the spine, external swellings of various sorts, etc., the diagnosis of the disease may be considered as already made out.

This prodromal period may differ very greatly in duration; it may last months or years; its symptoms always precede for some time those of compression of the cord; they are absent only in case intra-medullary symptoms are prominent, when the symptoms of pressure may be the very first to be noticed.

The latter, belonging to the second stage of the disease, consist at first of a *paralysis*, of more or less rapid development, usually in the form of paraplegia, more rarely in that of hemiplegia, and in the latter case usually developing into paraplegia. The first symptoms of paralysis are often preceded for a time by *paræsthesia* in the lower half of the body; tingling, furriness, sensations of burning or cold, girdle-sensations, etc., are described by the patient. The order in which the sensory and the motor symptoms appear depends on the direction from which pressure is first felt; those which first appear may exist some time before the other set follows. Those of the motor class, however, very soon become more marked than the other set; the preponderance of motor paralysis is quite a characteristic trait in the picture of myelitis from pressure. The *paralyzed muscles* are first *completely relaxed and softened*, the joints are relaxed, and obey every passive movement without resistance. At the same time there is considerable *increase of the reflex actions*, both cutaneous and tendinous, in the paralyzed lower extremities, except when the lumbar swelling is the seat of pressure; the slightest irritation gives rise to the fullest reflex movements, convulsive twitching, and the like, in grotesque contrast with the absolute motor paralysis.

The paralysis of the bladder and rectum is often delayed, but if there is any considerable compression, it is sure to occur; the symptoms are the well-known ones of involuntary discharge.

As the disease progresses, the muscles, at first quite lax, become by degrees *tense* and *rigid*, and are attacked by *twitchings*, or transient tonic spasms; *contractures* appear, at first temporary, afterwards permanent; the lower extremities continue in a permanent position of extension, which afterwards gives place to flexion. At the same time there is an *additional increase of reflex action;* the *tendinous reflexions* especially gain in intensity, every slight dorsal flexion of the foot produces the most active clonic movement of both lower extremities, often a convulsive tremor, etc. The nutrition of the muscles may remain intact for a time, or they may emaciate.

At this point the cases divide; those of a less severe sort remain for a long time of about the same severity; slight improvement gradually appears; the anæsthesia is the first to diminish; the functions of the bladder are better regulated; the power over a few movements returns, increases by degrees; in short, the improvement may go on step by step to full recovery.

In severe cases, however, the symptoms become worse; the paraplegia remains complete; bed-sores appear on various places, cystitis, fever, general marasmus follow, and amid indescribable sufferings the patient sinks into his grave.

Let us now *analyze the symptoms*, and attempt to refer them to the existent anatomical lesions.

The most constant and most important of the prodromal symptoms is without doubt that of *pain*. It has a great deal that is characteristic; it is confined to very distinctly marked nerve-root districts, being in the beginning often localized in a line or a point; it is neuralgiform, lancinating, and often (in connection with marked neuritis) rather of a burning character. It is very distinct in carcinoma of the vertebræ, where it occurs in severe paroxysms, especially by night, is soon beyond the control of narcotics, and gives the patient fearful torments. A variety of eruptions of the skin often appear in the district of the cutaneous pain.

Without doubt these pains should be referred to the mechanical irritation, and most especially to the consequent neuritis of the sensory roots; this irritation is brought about by the increase of the compression, the sinking together of the vertebræ, and the narrowing of the intervertebral foramina.

As all these matters may differ in each case, it is readily seen that the degree and extent, intensity, character, and commencement of the pain may differ greatly in individual cases. We refrain from entering further into the details.

It is without doubt to the same cause—mechanical and inflammatory irritation of the sensory roots—that the hyperæsthesia is due which we observe in many cases, found usually in the parts where the pain is distributed, and varying greatly in character.

A further lesion of the roots is indicated by the *anæsthesia* occurring in their district of distribution, which may assume the form of a zone, or be restricted to certain cutaneous nerves, or to quite isolated spots of skin, according to the number of fibres compressed.

The symptoms of *motor irritation* and *paralysis* in the initial stage are referable to quite analogous states of the anterior roots. Tremor, spasms, cramps, permanent uniform painless contractures occur here, and alternate with paresis or paralysis of certain muscles and groups of muscles, or coexist with them. The location of these disturbances varies according to the seat of lesion; they will be noticed at an early period if the compression acts upon the roots passing off from the cervical or lumbar swelling, because in that case considerable disturbances at once appear in the extremities.

The fact that the motor and the sensory disturbances are referable to nerve-roots occupying nearly the same level in the cord is one which has been observed in many spinal diseases, but is especially distinct in this.

If the compression and neuritis of the anterior roots becomes severe, the now complete paralysis of the corresponding muscles is followed by progressive *atrophy*, and with the atrophy there is very closely associated a corresponding *diminution and loss of faradic excitability*, which, as will be seen upon closer examination, is only a portion of the reaction of degeneration.

It hardly need be said that in these paralyses from pressure on the roots the reflex actions are always extinguished, and that this circumstance is not without value in the diagnosis.

In the second stage the symptoms of *motor paralysis* are the first and the most prominent. In fact, *paraplegia* is often enough the first symptom that indicates the severity of the disease. It may develop with more or less rapidity; a few hours or days are often sufficient, but usually weeks are required; the patient feels his legs growing heavier, he drags them in walking; the toes catch at the least obstacle; his knees double under him, and so walking and standing become impossible; at last there is a complete loss of all movement, even in the lying posture. The muscles remain perfectly lax, soft, and non-resistant under passive movement. Their electrical reaction is unchanged; their nutrition is at first quite unimpaired.

All this is perfectly intelligible when referred to the gradually increasing compression of the cord, with its accompanying myelitis; the slight initial symptoms of weakness may without hesitation be referred to compression, while a more rapid increase of paresis, a relatively rapid development of paralysis, is undoubtedly due to myelitis. At a later date, of course, the proportion of the influence of the two elements upon the origin of the complete paralysis cannot easily be estimated. As the disease is always circumscript at the beginning, the nutrition and electric reaction of those muscles whose nerves originate in the inferior (intact) portion of the cord will remain perfect until a descending affection of the gray substance threatens their functions.

As in most cases the compression takes place from the front of the cord (Pott's disease), it is readily seen that the motor disturbances will be the first to appear, and will for a long time outweigh the sensory.

In a minority of the cases, the compression is limited to *one-half* of the cord, leaving the other more or less intact. The paralysis then takes the form of *spinal hemiplegia* or *hemiparaplegia;* if the compression of the affected half is pretty complete, it may be associated with a crossed anæsthesia, and give rise to the characteristic unilateral lesion (see below, No. 14). Usually, however, after a longer or shorter time, the myelitis will extend over the entire transverse section, and complete the paraplegia.

The paralysis not infrequently begins in the form of a *cervical paraplegia, i. e.*, the upper extremities are first and completely palsied, while the lower are nearly or wholly free at first. This condition may be due to the fact that the lesion occupies the level of the cervical enlargement, and in this place first attacks the anterior roots for the upper extremities; in this form of palsy, muscular atrophy and absence of reflex actions form a characteristic feature. Or, if the lesion is situated in the upper part of the cervical cord, it may happen that the motor paths in the antero-lateral columns which belong to the upper extremity are at first exclusively attacked by the compression, and those for the lower extremities at a later period. This might perhaps be explained by supposing the former to lie nearer the surface of the cord than the latter, and thus to be sooner affected by pressure and myelitis. In this case the reflex actions are retained in the upper extremities also.

Finally, in a few rare cases, a so-called *recurrent paralysis* has been observed, that is, an upward extension of the paralysis above the point of compression; *e. g.*, in compression of the dorsal region, extension to the upper extremities. This is explained by the ascending myelitis, which often occurs, and in some cases by the very rare occurrence of ascending degeneration of the lateral columns, which may extend to the cervical enlargement (Michaud).

The *sensory disturbances* are not usually so marked as the motor, at least in the beginning. The development of paraplegic symptoms is often preceded for a varying period by *parœsthesiœ* (tingling, formication, burning, etc.), which may cover the whole body, and often continue without a break, or recur during the later course of the disease. They are sometimes due to compression of the posterior columns, and sometimes are signs of commencing myelitis in the posterior columns and the gray substance. The same cause may be assigned for the *pains*, which often appear during the later course of the disease; these have not the lancinating, neuralgiform character, but are rather an intense, diffuse sense of pain, burning, boring, pressure, etc., extending over the whole of the lower extremities. I have repeatedly observed them in the paraplegia of vertebral caries, and Michaud ascribes them to the myelitic irritation of the gray substance.

Charcot describes also an *abnormal irradiation* of pain and paræsthesia, a peculiar *dysœsthesia*, which is produced by various sensory irritations; it is a singular painful vibrating feeling in the lower half of the body, the same for all sensory impressions; this sensation must also be referred to the disease of the gray substance.

Symptoms of *anœsthesia* are most constant, though they may vary greatly in amount; the anæsthesia is usually incomplete, and not rarely presents a certain contrast to the severity of the motor paralysis; Courjon states that the sensibility is never quite destroyed in vertebral caries, though this certainly occurs in other forms of paralysis from compression. A *retardation of sensory conduction* is also often observed. All these symptoms are very simply explained by the different degrees of compression and of myelitis, by the greater or less implication of the gray substance, etc.

There is very little said of *vaso-motor* paralysis in the existing reports of cases; only Hawkins states that he has observed a constant elevation of the temperature of the paralyzed half of the body in a case of compression of the dorsal medulla. It is conceivable that the slow progress of the interruption of conduction in the cord may give the vaso-motor centres, situated in the lumbar region, time to develop sufficient activity to avoid serious vaso-motor disturbances.

Paralysis of the bladder is usually a subordinate feature of compression-myelitis, especially when seated above the lumbar enlargement; it then seldom appears early or in a severe form. This depends on the seat and severity of the lesion, the integrity of the centres in the cord, etc.; all shades of severity may be observed. The severest cases are those which accompany a pressure-myelitis of the lumbar medulla. The same is true of *paralysis of the rectum.*

The condition of the *reflex functions* is especially interesting; an exaggeration of their activity is one of the most constant symptoms of pressure-myelitis, provided that the portions of gray substance which preside over the reflex actions are not diseased.

The first thing which attracts notice is the increase in the cutaneous reflex actions, the lightest touch upon the skin provokes a vigorous reflex jerk; if the skin is pressed or pinched more strongly, active and powerful movements of the entire extremity are made, which often extend to the other inferior extremity, or, in their severest form, terminate in a convulsive twitching and shaking of the limbs, which may continue some time.

The *reflex actions originating in other parts* are also increased; every evacuation of the bladder or the rectum, the introduction of a catheter, etc., is accompanied by active, often painful, jerkings of the limbs. Irritation of the skin of the inner surface of the thigh, or of the urethra, has been seen to produce reflex erection of the penis.

The *tendinous reflexions*, however, are most markedly developed in such cases, and it is in myelitis from pressure that they are to be best studied. The lightest tapping of the patellar tendon or the tendo Achillis, the tibialis posticus, the tendons of the flexors of the knee, gives rise to reflex actions; the same is often accomplished from the periosteum of the bones and from various fasciæ; powerful pressure of the patella downwards produces reflex clonus in the quadriceps; the slightest dorsal flexion of the foot produces that clonic tremor of the lower leg, which in its highest degree extends to the whole leg, and then to the other leg, and terminates with an intense tremor of both legs, lasting some time.

Nothnagel has recently stated that all these reflex actions may, in favorable cases, be inhibited and arrested by a powerful pressure upon one of the great nerve-trunks of the lower extremities.

It can hardly be doubted that this increase of reflex activity must be referred to two circumstances: first, interruption of the conduction to the brain, which interferes with the function of the fibres for inhibition of reflex action; and secondly, hyperæmia and inflammatory stimulation of the gray substance. It is not at present possible to determine the part taken by each of these circumstances in increasing the reflex action.

It is interesting that a reflex action originating in a tendon, the centre for which is situated in the compressed region, may become extinct, and may afterwards reappear when the motility has returned and the pressure-myelitis is cured.

I have observed this in a case of kyphosis of the lumbar region, in which the upper part of the lumbar enlargement was evidently compressed. As long as the paraplegia continued, the tendinous reflexions from the ligamentum patellæ and the adductor tendons were completely extinct, while in the whole sciatic region they were considerably increased. After four months the paralysis disappeared, when the reflex actions from the ligamentum patellæ and the adductor tendons reappeared.

Another step in the disease of the cord is indicated by *rigidity of the muscles*. We have seen that at first they are completely relaxed. In a few days, however, or more usually in a few weeks, or still later, the muscles begin to be attacked by twitchings and slight spasms; they assume a condition of tension, oppose to passive movement a gradually increasing opposition, and at last *contractures* appear, at first transient, then permanent, which affect the disease in a characteristic way. Usually these are at first contractions in *extension;* the legs lie stretched stiff and straight side by side, the feet in the position of varo-equinus, the knees stiff and pressed together. At a later period, especially in vertebral caries, contractures in *flexion* always occur; the hip and knee-joints are strongly bent, the knees drawn up, the heels to the buttocks, the legs often crossed and locked. At first the contractures are comparatively easy to reduce, but return whenever the traction is taken away; afterwards they resist all external force.

It appears quite certain that these motor symptoms are referable to disease of the lateral columns, that they belong to the descending degeneration and sclerosis of the postero-lateral columns. They seem to be more marked in myelitis from pressure than in most other forms of the disease.

Trophic disturbances are not very prominent in myelitis from pressure, as long as certain sections of gray substance (in the cervical and lumbar medulla) are not directly affected. The nutrition of the muscles then remains intact a long time, and so does their electrical reaction; at the most we observe emaciation, caused by want of activity and by the general depression of the nutritive processes.

The case is otherwise when the lumbar or cervical enlargement is affected, or when the secondary myelitis of the gray substance extends upwards or downwards to those parts; the consequences, then, are rapid and great emaciation of the muscles, loss of faradic excitability, and the appearance of the reaction of degeneration.

In a few cases, *affections of the joints and skin* have been caused by pressure-myelitis.

Bed-sores are usually confined to the severer cases. The same is true of *cystitis* and other consequences of retention of urine.

After this review of the chief symptoms, we have to say a few words of the variation in symptoms which is due to the *different seat of the compressive lesion.*

The case is most complicated when the lesion occupies the *cervical part* of the cord. And here, again, we can form two subdivisions, according as the cervical enlargement, or the part of the cord above it, is the seat of compression.

In the latter case (compression of the upper part of the cervical cord), the disease not rarely begins with pain in the occiput, stiffness of the whole neck, obliquity of the head, inability to nod and turn the head, and so forth. The paralysis often begins and continues in the upper extremities (paraplegia cervicalis), while the lower are wholly or comparatively free. At a later time there is palsy of all four extremities. The reflex actions are retained in the upper extremities. Other symptoms, however, now appear, which are quite characteristic of this seat of lesion, and are due to implication of the nerve-paths there situated. Such are: *oculo-pupillary symptoms*, either paralytic myosis (from paralysis of the corresponding paths in the cervical medulla), or spastic mydriasis (from irritation of the corresponding paths), either unilateral or bilateral; *respiratory disturbance*, from implication of the respiratory paths; *gastric disturbances, repeated vomiting, difficulty of swallowing, continued hiccup*, to be explained by implication of the vagus, spinal-accessory, and phrenic. In many cases a striking and *permanent retardation of the pulse* has also been observed, down to 48–20 beats in the minute, accompanied by fainting-fits with complete cessation of pulse; these symptoms have been referred to irritation of the vagus (Charcot, M. Rosenthal); and finally, *epileptic attacks* have often been seen when the compression was situated at this point.

If the cervical enlargement is compressed, the initial symptoms of pain, anæsthesia, spasm, palsy, atrophy, are localized in the upper extremities; these are also the first to be affected, and the lower extremities follow later. The absence of reflex phenomena, and the atrophy which soon invades the upper extremities (see above, description of Pachymeningitis Cervicalis Hypertrophica, p. 221 et seq.), constitute decisive proof that this is the seat of disease. In this form, also, one or several of the above-named symptoms may appear (changes in the pupils, disturbances of respiration, retarded pulse, etc.), and complete the picture of the disorder.

The portion most frequently the seat of compression is the *dorsal region.* The symptoms are very characteristic: girdle-pains, intercostal neuralgia at various levels of the trunk, paraplegia up to the corresponding level, reflex actions in the lower extremities retained and increased, nutrition of the muscles and their electrical excitability intact, etc.

If the *lumbar region* is affected, the paralysis is confined to the lower extremities, the bladder, and rectum; the initial symptoms are localized in the lower extremities; reflex actions are absent, the muscles are permanently relaxed, are mostly atrophic, exhibit the reaction of degeneration, etc. The paralysis of the bladder and rectum is complete, and severe at an early period.

It scarcely need be mentioned, that the diagnosis of the location may be sustained and confirmed by various symptoms connected with the vertebræ—kyphosis, swelling, painfulness, etc.

If only *one lateral half* of the cord is compressed, the characteristic symptoms of Brown-Séquard's unilateral lesion appear: motor paralysis and hyperæsthesia, and increased temperature on the side of the lesion, bounded above by a zone of anæsthesia, while on the opposite side of the body there is (crossed) anæsthesia. (See below, No. 14.)

Course, Duration, Termination.—All depends on the nature of the primal lesion. In meningeal and intra-medullary tumors (see the section on these) the disease almost always goes on without stopping to its termination; death comes sooner or later, after months or years, and always amid the painful accompaniments of severe spinal paralysis—bed-sores, cystitis, fever, marasmus, etc.

The same is true in cases where vertebral carcinoma or other malignant tumors are the cause of compression, except that in these the fatal result is usually much more speedy.

But it is otherwise in *vertebral caries*, which is by far the most common cause of spinal compression. In most of these, the course is comparatively favorable. A good many perish by

ordinary process of severe paraplegia, bed-sores, pyæmic
, etc.; but the disease usually runs on more slowly to its
with remissions and exacerbations, often after an apparent
, if the vertebral disease is lit up afresh by an accident, a
ical injury, a severe cold, etc.

n *more favorable* cases, the paraplegia remains unchanged
uniform for a considerable time (two, five, to ten months,
to three years); bed-sores do not appear, or, if they do, are
ed. Finally, a slow improvement begins; a few movements
at times (for example, when in the bath) be executed by the
lyzed parts; then they are resumed permanently, grow
nger, the contractures disappear, sensibility improves, the
tion of the bladder is again controlled by the will, the excess
flex action diminishes. The patient then succeeds in stand-
and by degrees learns to walk, at first with crutches, then
a stick, and then without help. Thus he may be completely
red; but a number of months is always required for the pur-
. Such kyphosis as is present may remain without any
ge; evidently nothing has occurred but a diminution of the
lation in the spinal canal, by which a resumption of use, and
ir of structure, has been rendered possible.

he result is not always so completely favorable. Restora-
may remain imperfect; partial paralyses and contractures,
atrophy and anæsthesia, weakness and clumsiness in the
f the limbs, remain. Such persons are always in danger of
lapse, and any external injury may arouse the sleeping dis-

Diagnosis.

he entire group of symptoms, their beginning and succession,
lly enable us to decide with ease that a slow compression of
ord exists. Of characteristic importance are the initial root-
toms, which give warnings for a long time previous; and
wards, the more or less rapid occurrence of paraplegia with
ased reflex action, with muscles at first entirely lax, after-
s more rigid, etc.

he only point which usually presents difficulties is that of the cause of the compression. In many cases it will be quite impossible to ascertain the cause, as in small exostoses of the vertebræ, in meningeal tumors, etc.

In the ordinary cases, the points of diagnosis will be furnished by the external circumstances, those belonging to the primary disease as such, although these will not always insure a correct decision. We will give some instances.

Pott's disease usually presents the symptoms in their typical form, and the actual cause of them cannot be ascertained unless other symptoms, especially characteristic of spondylitis, are present—the gradual formation of kyphosis, especially if angular; painfulness of movements of spine; great tenderness of spinous processes when touched; severe pain when the electrodes of a galvanic current are placed close to the diseased vertebra (M. Rosenthal); abscesses of congestion; youth on the part of the patient, especially if he be scrofulous; in older persons, a previous traumatic lesion, etc.

In *vertebral carcinoma*, the violence of the initial pains is usually regarded as characteristic, but there are so many exceptions to the rule that this circumstance ought to be allowed, at most, only to establish a suspicion. The girdle-pains and other pains of excentric origin are often of fearful violence, and occur in paroxysms, especially by night. Great hyperæsthesia usually exists in the painful region, together with all other possible root-symptoms. The localization of the pain in the back close to the vertebral column is said to be characteristic (Gull). If, in addition, there exist a local pain in the vertebræ, progressive, round curvature of the column, symptoms of compression of the cord, an external prominence and general cachexia, the diagnosis becomes plainer, and the more so if a primary cancer can be shown anywhere else (as, for instance, in the breast)—the search for which should be a first duty. Yet it is possible to confound this affection, in its first stages, with almost any other one which compresses the roots.

In respect to *meningeal tumors* we have mentioned the most important points above (p. 262 et seq.); they are usually characterized by slow development, and also, to some extent, by the absence of every symptom of vertebral disease.

In regard to the *intra-medullary tumors* we shall speak more in detail farther on (see No. 18). It is doubtless extremely hard, in most cases, to distinguish them from cases of circumscript transverse myelitis of spontaneous origin. We merely remark here that the initial symptoms of irritation of the roots are usually absent, and that they open with symptoms of compression and myelitis caused by pressure.

The diagnosis of the rarer causes of compression of the cord—gout of the vertebræ, exostoses, syphilitic new formations, aneurisms, etc.—is made from the signs which are applicable to these diseases, though their uncertainty is well known.

Prognosis.

This follows easily from what we have stated, and depends in the first instance upon the fundamental disease. Carcinoma of the vertebræ, tumors of the meninges, exostoses, etc., are never recovered from. If they lead to compression of the cord, the prognosis is very bad, or absolutely fatal, and the most that can be hoped for is some modification of the duration of the disease, depending on the original cause and the greater or less rapidity of its progress.

Cases due to compression by syphilitic formations, perimeningeal exudations, and vertebral caries, are curable. The question whether restitution and repair of myelitis from compression is possible must be answered decidedly in the affirmative. Wherever the cause of compression is capable of removal, the cure of the paraplegia may be expected.

This seems to be far from uncommon in Pott's disease. Among six cases of paraplegia from vertebral disease which have come under my observation in the past year, five, including two adults, have been cured or decidedly improved, and only one has terminated fatally. A similar result is reported by a great many (Leudet, Charcot, Courjon, and others). Accordingly, the prognosis of such paraplegias is to be stated as comparatively favorable, with a certain reservation. A good recovery may be especially looked for in young, pretty well-nourished persons, in whom the disease of the bone has not assumed considerable dimensions, or led to large abscesses of congestion, and who also are not very scrofulous. Complete recovery, with the exception of the permanent deformity of the spinal column, seems to be a possible thing. In many cases, however, we must be content with an imperfect recovery.

Treatment.

Little can be said of this. In cases of severe lesion (carcinoma, exostosis, meningeal tumor, etc.) there is simply nothing to do, and we must be content with a symptomatic treatment for the relief of pain and other discomforts, and for prolonging the patient's life as much as possible.

The only object which repays treatment is formed by the cases of *spondylarthrocace;* a steady persistence in the use of rational means will often lead the disease to a more favorable course and gradual recovery. It is not our object here to enter into details regarding the treatment of vertebral caries, but only to cast a passing glance upon it.

First of all, the spine should be kept in repose as nearly as possible, for which purpose rest in bed for months on the face or back is necessary. Apparatus for support or protection of the spine may be very useful in cases where movement is unavoidable, or is required for other reasons, or, finally, when the improvement has made a certain degree of progress, and the spine requires still further care. The physician must be earnestly warned of the danger of those mechanical manipulations which are so often employed by ignorant orthopedists and bandagers for straightening the spine and relieving the curvature. It is quite certain that kyphosis is not usually the cause of paraplegia.

In general, a tonic treatment is to be recommended—good, abundant diet, such as is specially suited to scrofulous patients, fresh air, iron, quinia, cod-liver oil. For the disease of the bone itself, preparations of iodine are much in favor; iodide of potassium, or still better, iodide of iron, and externally, painting with tincture of iodine, or friction with strong iodine ointment. In proper cases, leeches or cups may be applied to the spine. Derivatives, vesicants, moxæ are great favorites. The white-hot iron has lately been repeatedly advised as peculiarly efficacious in Pott's disease (by Charcot, and others). Every two weeks, on each side of the curvature, from two to four eschars are to be made of the size of a half-mark piece (sixpence sterling), and passing through the entire skin. Strikingly good results have been observed from this treatment even in late stages of the disease. Brine-baths or warm sea-baths may support this treatment in many cases.

For the *myelitis from pressure* little can be done until the cause of compression is removed. In the cases, which have been before described, where the cause is not removable, we had better not undertake any treatment for the relief of myelitis.

In vertebral caries, also, we should do best to put off this branch of the treatment until the compression begins to diminish; but inasmuch as this point cannot be defined, and as the hope of doing good is likely to influence us at an early date, we shall usually find ourselves justified in employing the customary remedies for the relief of chronic and subacute myelitis (cf. below, Treatment of Myelitis). These include local bloodletting, derivatives, frictions with mercurial ointment, the exhibition of iodide of potassium, pencilling with iodine, etc.

When the process of regeneration has begun, there are various remedies which may possibly hasten it, as the continued use of nitrate of silver, iodide of potassium, iodide of iron, quinia, strychnia (used with the utmost caution), moderate hydropathic measures, and most especially the galvanic current. Ollivier (p. 481) gives a case, in which galvano-puncture was apparently followed by good results. I have acquired the impression from my own experience that a moderately strong galvanic current, applied in the stabile way to the point of lesion, decidedly favors the restoration of the spinal functions. I place the poles upon the spine, one above, the other below the seat of disease, and pass a weak current, at first in one direction and then in the other, for two or three minutes in all, once a day. I have never seen this do harm. Patience and perseverance are of course required for this.

The symptomatic treatment of the chief special symptoms (pain, spasm, atrophy, bed-sore, cachexy, cystitis, etc.) is governed by general rules.

When a cure is effected, the patient should be strictly warned against fresh injuries, which might bring on a relapse of his complaint.

6. *Concussion of the Spinal Cord—Commotio Medullæ Spinalis.*

Abercrombie, Diseases of the Brain and Spinal Cord. German tr. by G. v. d. Busch. 1829. p. 520.—*Ollivier*, L. c. I. p. 488.—*Leyden*, L. c. II. p. 92.—*Holmes*, System of Surgery. Vol. II. p. 238.—*Clemens*, Die Erschütterung des R.-M. und deren Behandlung durch Elektricität. Deutsche Klinik. 1863-65.—*Lidell*, On Injuries of the Spine, including Concussion of Spinal Cord. American Journ. of Med. Sci. Oct. 1864.—*Erichsen*, On Railway and other Injuries of the Nervous System. German tr. by Kelp. Oldenburg, 1868.—*Webber*, Recovery after Four Years' Paralysis, following Railway Injury. Boston Med. and Surg. Journ. July 18, 1872.—*Morgan*, Injuries of the Spine, Result of Railway Concussion. Med. Press and Circular. Jan. 1873.—*Scholz*, Ueber Rückenmarkslähmung und deren Behandlung durch Cudowa. 1872. p. 76.—*Erichsen*, On Concussion of the Spine, Nervous Shock, and other Obscure Injuries of the Nervous System. London, 1875.

Introduction and Definition.—Under the term, "Concussion of the Spine," we include those cases in which energetic traumatic influences (falls, blows, collision, etc.) have given rise to *severe disturbances of the function of the cord, without any considerable visible anatomical changes in the latter.* Slight changes, small capillary extravasations, etc., probably exist in such cases, but they do not seem to constitute the proper essence of the disease; for the most part, the anatomical change is quite negative, and we do not yet know what changes, if any, constitute the basis of the concussion proper.

In the preceding chapters we have considered cases in which severe traumatic injuries have given rise to coarse anatomical lesions—hemorrhages, crushing, hemorrhagic softening of the cord, etc. We have now to deal with cases in which this does not occur, and yet there are severe spinal symptoms. Whether these two classes of cases are different only in degree, and may gradually run into one another, we will not try to decide; but it does not seem very probable to us. We rather incline to the view that the concussion of the cord is a very peculiar kind of disturbance, and that more or less of it is usually present in those severer lesions, but concealed to some extent by the symptoms. We may here assume a similar relation to that which exists between concussion and contusion of the brain; the former being a condition unaccompanied by any certain and constant anatomical changes, and the latter involving crushing and destruction of brain-tissue.

We may here include with safety the conditions which have been named *shock of the spinal cord.*

The diagnosis is in many cases so uncertain, and the want of satisfactory evidence from autopsies so great, that the history of the disease is still surrounded by darkness. We shall, therefore, be as brief as possible, consistently with the great practical importance of the subject.

Etiology and Pathogenesis.

The most usual cause consists of a *fall of moderate height* upon the feet, buttocks, back, and less frequently on both arms outstretched and stiff. I have observed the symptoms in two ladies, produced by a fall on the ice and on a polished floor; a fall on the buttocks caused by slipping down stairs is often mentioned as a cause.

A blow from a heavy body in motion, striking the spine or trunk, acts in quite the same way.

A sudden shock to the whole body produced by the sudden cessation of a rapid motion—as occurs in a collision between vehicles—is a common cause of concussion; of late years *railway accidents* have played a very prominent part in such injuries, the rapidity of their movement essentially increasing the force of the shock. They sometimes seem to produce quite special forms of concussion, and have been carefully studied, especially in England, where the suits for damages against the railway corporations have given them a very great practical importance.

The action of any of these mechanical causes may be limited more or less to a portion of the cord, when the spinal column has been directly struck; but it may extend to a great part or the whole of the cord, if the shock has been indirect, or has affected the entire system at once. Severe symptoms do not always follow directly upon the accident; they are often delayed for weeks or months, and may not appear until some other injury has been received. We must then assume that the shock has only produced a certain tendency to disease of the cord.

All these mechanical causes may produce a more or less considerable lesion of the soft parts or the spinal column; which is quite a matter of accident.

Besides the mechanical causes, there are others capable of producing symptoms very like those of concussion of the cord, which we will not omit.

Clemens speaks of excessive *coitus*, conjoined with unusual excitement, or suddenly interrupted, or practised in the upright posture, as a not infrequent cause of a certain sort of concussion of the cord, said to be indicated by sudden weakness, collapse of muscular force, and subsequent severe spinal symptoms.

Violent mental excitement, especially fear or anger, is considered to play a similar part, and the paretic symptoms which often follow such excitement are referred to the cord; but the nature of the connection is quite obscure, and seems to us to be effected rather by congestion or myelitis, than by a change resembling concussion.

Finally, there is no doubt that a *stroke of lightning* often causes a general shock, in which the spinal cord participates, and which causes speady death. The person struck often recovers, but with paralysis, paraplegia, etc., for various periods of time, for which no anatomical basis has been found. A sort of concussion is, therefore, supposed to be experienced in such cases by the central nervous system, and in a few cases by the cord especially. We cannot form an exact idea of it at present.

The *pathological anatomy* of concussion of the cord is extremely imperfect.

In many cases which are examined at an early period, nothing at all is found in the cord, or at most one or two small unimportant extravasations of blood. Leyden reports a case which ended fatally in five days, in which the most careful examination disclosed no alteration of the cord.

In other cases anatomical changes are found, which are not severe enough to be considered causes of death; large and small clots, crushings, softenings of the cord in various places, etc.

In cases which terminate in death after a long time, chronic inflammatory changes may be found; it is supposed nowadays that chronic meningitis and myelitis, and various forms of gray degeneration and sclerosis, may gradually develop out of concussion; but this is not determined with sufficient precision.

It is, therefore, rather rash to entertain a decided opinion regarding the *proper nature of concussion of the cord.* It seems to be certain that the anatomical report is a negative one. The most common view, therefore, is that which supposes only molecular changes in the finer nerve-elements to have occurred, giving rise either to an immediate and complete functional paralysis of the latter, or forming the commencement of further disturbances of nutrition, which at a later time may result in degenerative inflammation. H. Fischer[1] has recently attempted to develop, in a complete form, another view of the cause of shock and concussion. He considers that which is known to surgeons as shock to be nothing more than a traumatic reflex paralysis of the vascular nerves; the concussion of the brain simply a shock localized in the brain, a traumatic reflex paralysis of the cerebral vessels. Scholz has applied this view directly to concussion of the spine.

We cannot see that Fischer's argument is convincing; it is hard to understand why, in so severe a shock, the vascular nerves alone should be paralyzed, to the exclusion of the other nervous elements; we rather believe that the latter are affected to at least as great a degree. This of course shakes the foundation of Scholz's application of Fischer's hypothesis to the cord.

For the present our opinion is that the *molecular disturbance is the chief element in concussion.* Such anatomical changes as may be present in individual cases are accidental and not essential adjuncts. It is perfectly evident that concussion of the cord must very often be complicated with contusion, hemorrhage within its substance, etc.

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[1] Volkmann's Samml. klin. Vortr. Nos. 10 and 27.

A very interesting statement has been made by Erichsen[1]—although it is not at present quite easy to interpret—to the effect that persons who are sleeping at the moment of a railway accident do not, as a rule, receive a concussion of the nervous system. Those sitting with their backs to the direction from which the shock comes, are the most severely injured. The same author draws an excellent comparison between the effect of a violent mechanical impression upon the cord, and the loss of magnetism in an iron bar which is struck with a hammer.

Symptoms.

The phenomena of concussion of the cord may vary very much ; there are many circumstances which influence it, such as the nature and the severity of the traumatic lesion, the degree of individual resistance, perhaps also neuropathic influences, the external circumstances as regards the care received, and the rest taken, with a variety of other matters.

In fully developed cases the essential point lies in the presence of a sudden, more or less complete loss of the spinal function ; if the concussion is local, this may be confined to the parts below the point of shock ; if diffuse, it may extend to the greater part of the body. We observe accordingly a more or less extensive paralysis and anæsthesia, coldness, cyanosis, weakness of the pulse, disturbance of respiration, retention of urine, etc. In some cases there is a gradual and imperceptible shading from slight weakness and relaxation to the severest palsy.

After some minutes, hours, days, or even weeks, movement and sensation return slowly ; often there is no other phenomenon developed until recovery is complete ; more often, however, a sort of stage of irritation follows, to which chronic inflammatory spinal disease may be added ; the latter may under some circumstances last a long time and result badly.

Not every case, however, begins with severe symptoms ; there exists a class of cases which ought, without doubt, to be included here, which, commencing with the most trifling symptoms, afterwards develop into a serious chronic spinal disorder.

The result is the production of a great diversity among the individual cases of concussion of the cord. For convenience we present here the following principal groups in outline :

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[1] On Concussion, etc. p. 120.

a. General and very severe symptoms at the instant of injury. Death in a short time. Severe form of shock.

The patient, after receiving some severe injury or other, is found completely paralyzed in all his extremities, with distinct anæsthesia, great prostration, often, though not always, with disturbance of consciousness, with involuntary evacuations. The pulse is very small, weak and slow, the skin cool and pale, or slightly cyanotic, the respiration disturbed, dyspnoic, etc.

In a few hours or days death occurs amid general prostration, increasing collapse, and paralysis of the respiration and circulation.

In this class must be included the cases of severe injury to the cord which end fatally in a day or two without any visible lesion (as crushing) which should necessarily involve death.

These disturbances are plainly due to a severe molecular concussion of the substance of the cord, whereby its internal nutrition is impaired and arrested.

b. Severe symptoms at the moment of receiving the injury. Cure in a short time. Slight shock.

Immediately after the accident the patient is found, usually in full possession of consciousness, complaining of severe and general pains in his body or in the lower half of it. The lower extremities, less commonly the upper also, are more or less severely paralyzed; usually there is anæsthesia also, but the latter is not always present, and often is but slight. The bladder is not always paralyzed. There are no symptoms of spasm. If such a case is seen within a moderate time after the accident, the reflex function, especially that connected with the tendons, may be found exaggerated, and the electrical reaction in the paretic parts may be increased or depressed.

In a few days improvement appears; the patient regains the power to stand and walk, but slowly, hesitatingly, feebly, and with tremor. The pains disappear; improvement makes rapid progress, and in a few weeks the recovery is complete. As an example of this form I repeat the following case in brief.

Johann Schaefer, æt. 55, day-laborer, four weeks ago fell a distance of twenty feet from a tree, landing on his feet and buttocks. He was not unconscious, but was paralyzed immediately, and had to be carried home. The following symptoms were there observed: violent general *pains in the loins and legs. Legs quite paralyzed* and immovable for about a week, when improvement gradually commenced, so that the patient can now walk a couple of steps. The *sensation of the legs was always good;* no anæsthesia was observed. Evacuation of the bladder always normal. Stool retained the first day, afterwards regular. The pains have disappeared by degrees, but the legs still tremble and are stiff.

Present condition.—The patient can scarcely walk two steps, and does it slowly, hesitatingly, dragging his feet, but without ataxia. Standing on the toes or on one foot is very difficult. Both legs tremble when he stands. Sensibility of the lower extremities quite normal. Cutaneous reflex functions retained. Tendinous reflex function strikingly active. No distinct atrophy of the legs. The electrical excitability of the nerves and muscles of the lower extremities is remarkably depressed, without qualitative change. Sphincters and upper extremities quite normal. No change in the back and vertebral column. Region of sacrum somewhat sensitive to pressure.

The galvanic treatment (to the spinal column and legs) was wonderfully successful; after a few sessions the patient was able to walk quite well, and was discharged cured after twenty-two (daily) sessions. The electrical reaction was again almost normal.

c. Severe symptoms at the first; followed by a protracted illness of some years' duration; recovery in most cases.

Shortly after the accident, the patient displays great weakness, which rapidly increases to a paralysis of various extent, often embracing all the extremities. With this are associated acute pains, more or less diffuse, often chiefly located along the spine, in the back of the neck, and loins. Paræsthesia occurs; cutaneous anæsthesia is not usually very marked. Retention of urine and retardation of pulse occur. In many cases an implication of the brain is indicated by unconsciousness and vomiting at the first; or by an increased irritability of mind afterwards.

Slow and gradual improvement occurs; great weakness of the extremities, slight atrophy of the muscles, acute pains and great sensitiveness remain. The extremities are cool and livid, the vertebral column is painful to pressure, and often excessively sensitive. The patient is obliged to learn to walk by slow degrees. After a long time—often years—a condition which approaches recovery is attained; but the patient always remains irritable and sensitive, and has to guard against injury. The following may serve as an illustration:

iss X., aged twenty, in April, 1872, slipped and fell on a polished floor, com- own on her seat. She immediately felt a severe *pain in the back of the neck oins*, with *great weakness*, but was able to go into another room. In a quarter hour she *vomited*, the *pains increased*, there was *severe paresis* of the entire , so that she could not even raise her head. All efforts to move were extremely ful, and she was excessively sensitive to light. Vertebræ very sensitive to pres- ; numbness of hands and feet, oppression of the chest, weak and slow pulse; were the leading symptoms at first. Urine retained only during the first day. e third month she begins to be able to lift her head a short time; the feelings æcordial distress disappear; afterwards the movements of hands and feet are vered. At the beginning of September the patient can walk a few steps, if orted. Improvement very slowly progressing.

t the beginning of June, 1873, I found the patient a fresh, healthy girl in earance, but with very excitable nerves. In *walking*—for which a slight support quired—a marked *slowness and difficulty in performing the motions* strikes the rver. Her back seems weak, and vacillates in various directions. In a few tes she sinks upon her knees, and is forced to sit down. *Standing* is accom- ed tolerably well for a considerable time. *Sitting* without a support for the is possible only for a short time. No ataxia. Given movements are easily uted with the legs, but without force. Arms and head quite free from symp- . No difficulty with the bladder, no sense of constriction, no palpitation of eart. *Sensibility* normal everywhere; slight numbness is said to be felt occa- lly in the soles. Spinal column straight, easily moved. The spinous pro- s of the cervical and upper dorsal vertebræ, and those of the lumbar vertebræ, ery sensitive to pressure.

alvanic treatment is carefully commenced, and is followed by a rapid and ressive improvement. By the middle of August the patient can walk quite ely without a stick. She then makes use of the cold-water treatment in zerland, from which she returns with fresh improvement. Another five weeks' e of galvanic treatment is also attended by good results. The patient was cured in the course of the year 1874, and in 1875 was married.

d. Very slight symptoms at the beginning; a severe progres- e spinal disease develops after a longer or shorter time. Re- l doubtful.

At the first moment—*e. g.*, if a railway collision—the symp- s are very insignificant. The patient has a sensation of hav- been severely shaken, a momentary weakness, perhaps some fusion of mind, but soon recovers, picks himself up and walks nt, dismisses apprehension from his mind, and goes on his ney.

n the next day, or several days later, or even after weeks and months, more threatening symptoms set in, perhaps preceded for a considerable time by very slight and unnoticed premonitions. A general depression of strength, sleeplessness, slight mental indisposition, tendency to shed tears, etc., may be noticed; the patient cannot attend to his usual business; pains appear in the back and limbs, and gradually increase.

Out of these symptoms a group gradually develops, which is very far from being identical in given cases, but which in general presents the following as its chief features: increasing weakness of the legs, which may reach different degrees of severity; the gait is uncertain, straddling, stiff, and dragging; uncertainty in standing, indications of disturbed co-ordination are often present. Stiffness of the back and the general attitude. Back painful, especially when moved; some of the spinous processes extremely sensitive. Girdle-sensation, paræsthesia of all sorts, anæsthesia of various degree and location, and often hyperæsthesia. Weakness of the bladder, diminution and loss of sexual power. Impaired general nutrition, pale sallow complexion, changed expression of countenance. Marked atrophy in certain muscles and groups of muscles, often quite extensive. Disturbances of circulation, bluish complexion, cold extremities, etc.

With these are usually conjoined symptoms which point to a disturbance of the cerebral functions; broken, poor sleep, timidity and irritability, weakness of intelligence, impairment of memory and of power to work, change of character, constriction of the head, increased irritability of the senses, etc.

These are essentially the marks of a very slow meningo-myelitis, associated with more or less considerable disturbances of the cerebral functions.

The subsequent course of the disease usually fluctuates a good deal. Periods of apparent improvement and comparative health alternate with those of downward progress. On the whole, a gradual loss usually occurs; a favorable result is seldom seen; but cases occur in which, after a very long time, the disease has considerably improved, or at least has ceased to make progress.

This category is largely composed of the cases which Erichsen has so admirably described; they have been observed more frequently of late, especially as a consequence of railway accidents, and have acquired a great practical importance in connection with the latter (*railway-spine* of the English). They may, however, equally well follow other severe concussions of the body, and especially of the back. Clemens describes a similar case, in which, after a fall from a scaffolding, atrophy and paralysis began to appear three-quarters of a year after the accident. The two last observations by Scholz are excellent instances of this form of concussion. There are various cases of progressive muscular atrophy originating in surgical injury which might be included here.

Diagnosis.

It is a matter of no small difficulty to establish a concussion of the cord with certainty, since the symptoms, especially at the beginning, possess a very close resemblance to those of slight hemorrhages or contusions of the cord.

The entire group of concussions of the spinal cord is still somewhat doubtful and undefined, and by many is only retained in order to serve as a receptacle for certain cases which cannot otherwise be easily interpreted. We shall make an attempt at a stricter definition.

The distinguishing character of concussion is the fact that, in consequence of some one of the above-mentioned causes (especially a traumatic lesion), severe disturbances of the functions of the cord occur, while at the same time the entire course of events shows the absence of any severe anatomical lesion, such as often actually follow such accidents.

The case may then take one of two forms: either (1) severe disturbance quickly appears, most severe immediately after the injury, and is followed in a comparatively short time by improvement, disappearance of the grave symptoms, until recovery is complete; or (2) no symptoms appear at first, or at most only trifling ones, the functions of the cord are comparatively free, the idea of a severe anatomical lesion of the cord seems inadmissible, and yet after a longer or shorter time severe and increasing disturbances do follow, which indicate a profound affection of the cord. In both cases we shall be compelled to assume the existence of molecular changes due to the traumatic lesion.

Either class, however, may give rise to mistakes.

Cases of the first class may be mistaken for crushing and contusion of the cord, with hæmatomyelia and hæmatorrhachis. The symptoms of all these may be very much alike at the beginning; but it is not impracticable to draw the diagnosis. Concussion agrees with crushing and tearing and with hæmatomyelia, in the severity of its initial symptoms, its paralysis, etc.; but its course is much more rapid and favorable. This point is entirely decisive. When, therefore, an apparently severe paraplegia comes to a favorable ending in a few days or weeks, without bed-sores, etc., we should assume the existence of concussion. The rapidity of recovery and the favorable termination belong to hæmatorrhachis and to concussion; but the initial symptoms are different, being usually more severe in concussion. In hæmatorrhachis the preponderant symptoms are those of pain and spasm; the paralysis is slighter; in concussion the opposite is usually the case.

The hypothesis of a concussion may be favored by the following circumstances: distribution of the paralysis over the entire spinal region *without* the corresponding disturbance of respiration and the rapidly fatal result which are the regular consequences of crushing of the cervical region; paleness and coolness of the skin, small, retarded pulse, absence of dislocation or fracture of the vertebræ, absence of pain and stiffness of the back at the commencement, etc.

Cases of the second class are not essentially distinct from those of myelitis or myelo-meningitis with a slow beginning, and are to be known only by the cause—the immediate and unquestionable connection of the symptoms with some traumatic or similar cause. The concussion should then be regarded only as the cause and starting-point of an organic disease.

We believe that the points of view we have taken will enable the reader to form at least a more correct judgment and a clearer definition in many cases of concussion of the cord. Much remains to be done in this respect; the first thing consists in collecting accurate reports of cases, avoiding, more carefully than has hitherto been done, the intermixture of other sorts of lesions.

The diagnosis will have to remain very obscure in many cases; it will of course be the most difficult, where with the concussion there exists at the same time some severe lesion, as crushing or bleeding of the cord. In such cases it will often be impossible to make an accurate diagnosis; but in many a careful sifting of evidence will perhaps succeed in distinguishing the two classes of injury. The diagnostic evidence will here consist in the disappearance of the symptoms of concussion from a part of the body—a reduction, as it were, of the functional disturbance to that point where it is properly commensurate with the anatomical lesion.

Prognosis.

In the severest forms of concussion of the cord, known as shock, the prognosis is always very grave. The lighter cases of this sort mostly recover; if improvement rapidly occurs, and good care is taken of the patient, the prognosis will be quite favorable. It is, on the whole, not very bad, when compared with the severity of the brief initial symptoms.

At all events, cases with severe initial symptoms seem to be the very ones which warrant a favorable prognosis as compared with those whose development is slow (Erichsen).

But even in protracted and sluggish cases, the prognosis is not absolutely unfavorable. If distinct myelitic or meningitic symptoms appear, the prognosis must be made as for these diseases; but even in the latter case, an attack which originates from concussion in a comparatively well person admits of a more favorable prediction than one of spontaneous origin. But when the improvement ceases, when after one or two years of rational treatment it makes no further progress, recovery is hardly to be expected.

If there is a severe anatomical lesion along with the concussion, such a lesion determines the prognosis when the danger of shock is over.

Treatment.

According to the form assumed, the treatment must vary.

In many cases the most urgent indications are those for *treatment of shock*. We must first make a most careful examination, must observe the pulse, the respiration, etc. A quiet and easy place to lie, warmth to the body, covering with warm clothes, rubbing the skin, are the first things to attend to. Then, in most cases, *stimulants* must be given in full doses; we should select, according to circumstances, wine, coffee, tea, hot spirit and water, cognac, and the like; or such drugs as aromatic spirit of ammonia, ether, musk, camphor, etc. In severe and threatening cases, strong cutaneous irritation is indicated, large sinapisms and vesicants, the faradic brush, etc. It must be decided by further experiments whether the subcutaneous injections of strychnia employed by Leyden will be serviceable.

Bloodletting, formerly very popular, must always be used with care in such cases; we may employ it in certain cases of robust persons, of full habit of body, if their pulse is strong, the temperature normal or increased, the spinal column decidedly painful at one point, or if we suspect some anatomical lesion, etc. We shall rarely have reason to use general bloodletting; the local will usually suffice.

In the second place, we shall have to treat *symptoms of reaction*. Here, too, absolute rest in a suitable position is required; if the patient does not bear lying on the side or face, he may lie on his back upon a couch tilted down at its foot (Erichsen). According to the violence of the symptoms, we shall then have to use the ordinary remedies for hyperæmia of the cord, for slight meningitis and myelitis—cold, moist or dry cups, derivation to the skin and intestine, ergot, iodide of potassium, etc.

A careful *watching of the period of convalescence* is necessary after these symptoms have passed away. The patient must carefully avoid all injurious things; especially, he must strictly abstain from bodily and mental excess of work, sexual excesses or excitement, colds, unusual jarring of the body (as by long drives, driving in bad roads, etc.); and he must be careful to get enough sleep. Recovery can often be favored by careful friction with cold water, by moderate use of the galvanic current (ascending and stabile through the spinal column), or peripheral faradization; by the careful use of chalybeate spring-baths abounding in carbonic acid (Cudowa, Schwalbach, etc.); also, by the internal use of tonics, iron, quinia, cod-liver oil, etc. Strychnia should not be resorted to until all the symptoms of irritation are past. Patience is necessary, as many of these cases last a desperate while.

It remains to speak of the treatment of the *sequelæ*, tedious and often severe, which follow so many cases of concussion of the cord. In most of these cases the treatment of chronic myelomeningitis will be appropriate. Quiet, and a well-ordered life, are of the first importance, and after this we may attend to the administration of special remedies, according to the usual principles—of which the chief will be the galvanic current, derivations to the skin, and iodide of potassium. Erichsen praises the efficacy of a combination of corrosive sublimate and quinia. The preparations of strychnia and iron will not be called for until a later period, when things have taken a favorable turn. The mineral springs are of especial importance in these cases; a proper selection is difficult to make, in the present state of our knowledge. Thermæ, especially the hotter ones, seem decidedly injurious, while moderate and careful cold-water cures seem distinctly useful. Scholz praises the Cudowa springs as the chief resource in most cases of concussion of the cord; he states the indications somewhat thus: Cudowa is indicated in all pure, uncomplicated cases of concussion; in the later periods it is especially indicated when there are few inflammatory symptoms, even in marked paralysis and anæsthesia. Cudowa is nearly useless for distinct meningitis.

In all circumstances, the treatment of these severe and prolonged cases requires great care and skill.

7. *Functional Irritation of the Spinal Cord—Spinal Irritatation.*

Stiebel, Rust's Magazin. XVI. p. 550. 1823.—*C. Brown*, On Irritation of the Spinal Nerves. Glasg. Med. Journ. No. 2. May, 1828.—*T. Pridgin Teale*, A Treatise of Neuralgic Diseases dependent upon Irritation of the Spinal Marrow, etc. 1829. —*Hinterberger*, Abhandlung über die Entzündung des R.-M. u. s. w. Linz, 1831.—*W.* and *D. Griffin*, Observations on Functional Affections of the Spinal Cord, etc. London, 1834.—*Ollivier*, l. c. II. p. 209.—*Stilling*, Physiologische und pathol. etc., Untersuch. über die Spinalirritation. Leipzig, 1840.—*Tuerck*, Abhandlung über Spinalirritation. Wien, 1843.—*G. Hirsch*, Beitr. zur Erkenntniss und Heilung der Spinalneurosen. Königsberg, 1843.—*Eisenmann*, Zur Spinalirritation. Neue med.-chir. Zeitung. 1844. No. 1.—*A. Mayer*, Ueber die Unzulässigkeit der Spinalirritation als besondere Krankheit. Mainz, 1849.—Die Lehre von der sog. Spinalirritation in den letzten 10 Jahren. Archiv der Heilk. I. 1860.—*Romberg*, Nervenkrankheiten. 3. Auflage Bd. I. p. 184. 1853.—*Wunderlich*, Handb. der Pathologie und Therapie. 2. Aufl. III. p. 28. 1854.—*Axenfeld*, Des névroses. Paris, 1863. p. 284.—*Radcliffe*, Reynolds' System of Medicine. II. p. 640. 1868.—*Beard* and *Rockwell*, A Practical Treatise on the Uses of Electricity. p. 350. 1871.—*Hammond*, l. c. p. 397. 1873.—*Leyden*, l. c. II. p. 3. 1875.

Introduction and Definition.—A good deal of change has taken place in the views entertained respecting the existence, the pathological position and significance of the group of symptoms which has been so well known by the term "spinal irritation" since the time of Brown (in 1828). Sometimes greatly overrated, its importance and frequency exaggerated beyond limit, and used as a common term for many most heterogeneous forms of disease in which pain of the back and sensitiveness of the vertebræ happened to be present, spinal irritation has been considered one of the commonest of diseases; while again, at the time when pathological anatomy was made the sole judge of everything, it was entirely denied recognition, or regarded as at most a frequent and rather meaningless symptom, so that it has almost passed from the memory of the present living generation of physicians.

No one, however, who has had much practical experience and who understands how to observe, can have failed to see that there is a considerable number of cases which by no means deserve to be confounded with hysteria, as has commonly been done; and which, on the other hand, do not agree with the other forms known to us, especially the ordinary spinal complaints, while they exhibit a sufficient mutual resemblance and agreement.

Such cases occur chiefly in the female sex. They are characterized by a great irritability of the sensory functions, with motor weakness and debility, in which one of the most constant symptoms is pain in the back and great sensitiveness of many spinous processes to pressure. In these cases the group of symptoms, as a whole, and the general course, unite to exclude with certainty any coarse anatomical lesion of the nervous system.

These diseases, which are distinguished by the great inconstancy of the symptoms, and a great variety in the localization and the apparent nature of the case, but which possess certain essential features in common, we will name "spinal irritation," and we justify the use of the term by claiming that the disease it represents is sufficiently characteristic in form. It must be admitted that the term is only a symptomatic one, as long as the pathological anatomy is so completely in the dark.

We would state plainly, that the idea of spinal irritation involves its own complete series or group of symptoms; and that all other known forms of disease, especially all organic diseases and tangible anatomical lesions, must be excluded. Thus we throw out all those cases which have so much confused our ideas of spinal irritation, in which the simple presence of spinal pain and tenderness have been supposed to prove the existence of spinal irritation. Spinal pain occurs in numberless diseases—hysteria, intermittent fever, many affections of the thoracic and abdominal organs (compare Tuerck's instructive presentation); but this does not imply that spinal irritation is present in these diseases. In hysteria, the entire series of symptoms often occurs, and so do, not infrequently, all other possible neuroses (intercostal neuralgia, migraine, spasm of the diaphragm, etc.). Nevertheless, spinal irritation does occur in an isolated form, and deserves separate consideration. It is the duty of diagnosis to establish in each case the independence or the secondary nature of the disease.

We have no more right to refuse to spinal irritation the claim to a separate existence, simply on the ground of the want of a known basis of anatomical lesion in the cord, than we have to do the same in acute ascending paralysis, in tetany, and many other diseases which betray an equally imperfect knowledge of pathological anatomy.

At all events, we believe that in the statement we are going to make, we shall present forms of disease with which practitioners are well acquainted, and for which neither the diagnosis of "hysteria," nor general "nervousness," nor any known anatomical form of disease, sufficiently accounts.

Etiology.

The *female sex* is *very much predisposed* to the disease. The number of women suffering from spinal irritation is far greater than that of men; yet the disease occurs in men also. *Youth* is also decidedly liable; very much the greater number of cases occur between the fifteenth and the thirtieth year. Finally, *hereditary neuropathic tendency* plays a very considerable part.

Among the *direct causes* it is usual to enumerate everything which excites and weakens the nervous system, and depresses its power of action. This includes strong psychical impressions, great excitement of the feelings, fright, grief, care, unfortunate love, violent passions, etc.; also excessive bodily exertions, severe marches, watching by night, work by night, etc.; in like manner, great sexual excitement and excesses, onanism in excess, continued and frequent sexual excitement without gratification; and finally, bad food, imperfect formation of blood, exhausting diseases, losses of blood and fluids. All these things may produce spinal irritation.

Intoxication with alcohol or opium, traumatic agencies, cold, etc., are also named as occasional causes.

At the time when every patient with pain and tenderness of the spine was considered to have spinal irritation, numberless diseases of the peripheral organs, and especially those of the intestine and uterus, were considered as giving rise to a *symptomatic* form of spinal irritation, as it was called. Such a thing is now no longer spoken of.

As we do not yet know what takes place in the cord in cases of spinal irritation, and as the pathological anatomy of the disease does not at present exist, it is hard to form a reasonable idea of the nature of the action of all these causes. We gladly omit all statements regarding the pathogenesis of the disease.

Symptoms.

The development is usually gradual. Slight pain and discomfort in the back appear, especially between the shoulder-blades—at first only upon unusual occasions, during excitement or fatigue; by degrees they become more permanent and require less and less to produce them. To these are added all kinds of excentric pains, increased nervous irritability, loss of general power, etc., and all this increases until the disease is fully developed.

Often, however, the development occurs quickly—in a few days, especially when very powerful influences have acted upon predisposed persons.

The disease then presents the following general aspect:

The patient is oppressed by a more or less troublesome sense of illness; a general malaise, increased psychical irritability, has seized upon her. In most cases she complains especially of *pain in the back*, situated in various spots, but most frequently between the shoulder-blades, next in the back of the neck, less frequently in the loins. It grows more severe when any movement or exertion is made, and in the exacerbations of the disease.

An examination usually discovers at the spot mentioned a *great sensitiveness* to pressure, tapping, the passage of a hot sponge, electricity, and other irritations. This sensitiveness may be so great that the lightest touch calls forth loud expressions of pain, the weight of the clothes becomes intolerable, and leaning the back against anything impossible. The skin of the affected portions of the back is usually very hyperæsthetic, but the spinous processes themselves are usually very sensitive to pressure. The degree and character of the pain differ greatly in individual cases; the pain is commonly described as a more or less severe sense of aching, which often lasts a considerable time beyond the effect of the irritation. Hammond describes, in addition, a deep-seated pain of the back, which, he says, is produced by pressure on vertebræ which are not sensitive, by movements of the spinal column, by standing, etc.

To this are added a crowd of other symptoms; but what most troubles the patient is the *pain* felt in various parts of the body:

neuralgiform pains, now in the upper extremities, or the occiput, or face; now in the trunk or viscera, assuming the form of various visceral neuralgiæ; again in the lower extremities, pelvic region, bladder, or genitals; pain often of great violence and severity, sometimes fleeting, sometimes more permanent, and often brought back by slight causes.

With these pains *paræsthesiæ* are often connected; tingling, formication, a sense of burning and heat, often of cold also; but these are less prominent. The same is true in a still greater degree of actual *anæsthesia;* it seems to be very seldom observed.

Marked *disturbances of motility* are regularly observed—above all, great weariness and exhaustion upon slight efforts; the patient has lost all endurance in walking, can walk but a little way, and presently not at all, on account of the intolerable pain the act occasions. Most patients therefore find it agreeable to lie on the back, and usually continue thus. Manual occupations, such as knitting, sewing, piano-playing, writing, etc., are more and more restricted, and at last are quite suspended, chiefly owing to the pain produced in the back or limbs. No real paralysis usually exists; all movements are possible, but provoke violent pains; and there is no power of endurance. The nearest approach to palsy consists in a moderate general paresis, occurring in but few cases; proper paralysis is not one of the symptoms of spinal irritation.

On the other hand, much is said of *spasmodic* symptoms; fibrillary twitchings, spasms of some muscles, choreoid movements, singultus, etc., are often observed. Even permanent contractures, epileptic attacks, etc., are said (probably without truth) to have been observed as consequences of spinal irritation.

Vaso-motor disturbances are also very frequent; most patients exhibit an abnormal irritability of the vessels, and easily turn red or pale; most of them suffer from marked coldness of the hands and feet, which are often of a bluish, cyanotic color.

Functional disturbances of the vegetative organs, of a great variety of forms, are also very common; eructations, nausea, even vomiting occur; palpitations of the heart are very frequent; disturbances of breathing, spasmodic cough, etc., are less common; while vesical spasm, increased desire to urinate, abundant discharge of pale, clear urine, are more frequent, but actual palsy of the bladder and rectum does not seem to occur.

Finally, a symptom, which seems quite a regular one, is that of *increased psychical irritability* and depression, with more or less *sleeplessness;* there is often some dizziness, noise in the ears, inability to read continuously, owing to the appearance of muscæ and disturbances of vision, etc.

The physiognomy of the disease is thus seen to be very complex. In fact, individual cases also differ greatly. We may try to divide them into three classes, according as the symptoms point to the upper, the middle, or the lower parts of the cord as the chief seat of suffering.

If the *upper portions* are principally affected, the pain of the back and spinal tenderness are chiefly localized in the cervical vertebræ. The prominent symptoms are those referred to the head, giddiness, sleeplessness, disturbances of the senses, pains in the occiput, and pains in the district of the brachial plexus; nausea, vomiting, palpitations, hiccup, etc., are not rare; motility in the upper extremities is usually impaired.

If the *dorsal portion* is affected, the chief symptoms in addition to those in the spine are intercostal neuralgia, gastralgia, nausea, dyspepsia, etc.; the lower extremities usually take a large part in the disturbances of motility and sensibility.

If the *lumbar portion* is chiefly affected, the leading symptoms are neuralgia in the lower extremities and the pelvic organs, spasm and weakness of the bladder, cold feet, weakness of the legs, etc.

A certain *generalization* of the disease is not uncommon, when the spine is painful in several places, often quite generally, and the disease is complicated by all kinds of peripheral symptoms.

Course, duration, termination.—We have already described the way in which the disease begins. Its *course* is usually very fluctuating. Improvement and relapses alternate in the most irregular way; the chief symptoms and the spinal tenderness are sometimes felt in one place, sometimes in another; a relapse often occurs without any visible cause, and so does improvement; it is here that we must be most on our guard against illusive successes.

Many cases run a comparatively acute course, grow rapidly worse, and as rapidly improve and recover.

In most cases, however, the disease is extremely slow and chronic, and its *duration* is stated in months and years; there are some patients who suffer more or less from occasional attacks all their life, and who are exposed to a relapse on the slightest occasion.

Nevertheless, a *cure* may be regarded as the rule; if proper measures are taken and the causes avoided, this may be expected in the majority of cases. Much patience will doubtless be required, and the many relapses may often greatly prolong recovery.

Whether spinal irritation may in bad cases result in the development of severe spinal diseases, does not seem to us sufficiently ascertained. The observations are almost all of an elder date, and give no sufficient guaranty against the confusion of the first stages of severe spinal lesion with functional irritation of the cord. This question, therefore, can only be decided by further careful observations. The entire doctrine of spinal irritation requires a renewed revision by means of careful and critically sifted clinical observations.

Until this is done, we shall not be in a position to entertain a better founded opinion upon the *nature of spinal irritation* than we now possess. We can scarcely doubt, it is true, that the structures within the spinal canal are the proper seat of the disease, and the entire list of symptoms makes it most probable that the cord itself is in a condition in which it performs its functions badly. The assumption that the meninges are the first to be affected, and the nerve-roots and the cord suffer secondarily, has little support.

But we possess no direct observations to show what sort of changes occur in the cord. The pathological anatomy of spinal irritation does not exist; the few autopsies which we possess are not always uniform, and some of them certainly do not relate to cases of this disease. We are therefore thrown back upon guesses and hypotheses regarding the real nature of the change in the cord in spinal irritation. Such hypotheses are numerous, but we have not the time to spare for them. The contradictory views that are entertained by authors are well illustrated by the fact that Ollivier and (in part) Stilling also refer spinal irritation to hyperæmia of the cord, while Hammond affirms with the fullest conviction that it is due to anæmia of the cord, and especially anæmia of the posterior columns, and that the ultimate cause for it may possibly exist in the sympathetic (vaso-motor) system; Beard and Rockwell assume at one time anæmia, at another hyperæmia as a cause, while Hirsch and many other writers see in spinal irritation nothing but a so-called dynamic disease, a functional disorder of the cord, without organic change in it—an irritation which may be due to very various causes.

All these opinions may be defended and attacked with powerful reasons, but we need not try to sit in judgment on them, as the conclusion could only be that we know nothing definitely at present. The most probable seems to us to be a purely functional disturbance of certain nervous elements of the cord, in company with which hyperæmia and anæmia of the cord may probably appear when the vaso-motor paths are reached by the disturbance; but this whole question, it seems to us, awaits a solution.

Diagnosis.

It will not be very hard to recognize spinal irritation when the entire group of symptoms as above given is present; when the pain in the back and the sensitiveness of the spine are accompanied by many changeable excentric symptoms, motor weakness, great psychical irritability, or marked paralysis or anæsthesia; when no organic changes exist, and a remarkable disproportion between the intensity of the subjective symptoms and the objective can be demonstrated; and where, finally, great fluctuations in the course of the disease are observed.

We ought not to try to settle the diagnosis too soon, nor should we acquiesce in it until, after careful examination and estimation of all circumstances, the other possibilities have been excluded. In doing this we should bear in mind the following:

The diagnosis from *hyperæmia* of the cord is so difficult, that cases were formerly often mistaken one for the other. The long duration of the disease will furnish the most important argument in favor of spinal irritation; in severe hyperæmia, paralysis is rarely absent. Hammond recommends as a test remedy a subcutaneous injection of strychnia, which is believed to do good in spinal irritation, and harm in hyperæmia.

The distinction from *meningitis spinalis* will also often be difficult. But in the latter the stiffness and painful tension of the muscles of the back, the pain in the spine, which especially occurs during movement, the fever, late paralyses, etc., may furnish very useful diagnostic points.

Meningeal tumors in their first stage, among the primary symptoms of which are found pain in the back and excentric neuralgia, will best be known by the stability of the symptoms, by their permanent localization in well-marked nerve-paths, and, at a later stage, by the paralytic symptoms.

The diagnosis from *myelitis* will usually be made soon. In this disease only deep pressure on the spinous processes is painful; there is no circumscribed cutaneous hyperæsthesia in the vertebral region, but there is the girdle sensation, very early and marked anæsthesia and palsy, vesical paralysis, often painful contractures and spasms, which are absent in spinal irritation. The unfavorable termination of the disease, the absence of the general nervous condition so common in spinal irritation, are likewise in favor of myelitis.

The distinction from *hysteria* will be impossible in many cases, as both diseases possess many similar features, and spinal irritation not rarely occurs in connection with hysteria. The specific hysterical symptoms of globus, general spasms, definite forms of paralysis, etc., do not belong to spinal irritation; and the peculiar mental traits so characteristic of hysteria—whimsical obstinacy, irritability, etc.—are also absent. A full consideration of the circumstances in each case will often furnish some decisive diagnostic points, while in other instances we shall have to admit the existence of both diseases together.

From *neurasthenia spinalis*, which is described in the following section, and which has an undeniable general resemblance to spinal irritation, the present disease differs by the fact that symptoms of sensory irritation preponderate, that extreme sensibility of the vertebræ is present, and that the disease occurs chiefly in the female sex. (See the diagnosis of spinal nervous weakness.)

The marks which distinguish spinal irritation from vertebral caries and other coarse lesions of the spine need not be stated here.

Prognosis.

This is generally held to be favorable, although such is not always the case. In all circumstances we should remember that the disease is usually chronic, may last many months and years, and relapses are very frequent.

Life is in no danger; but most of the patients are doomed to a long and tedious illness, they are cut short in all their enjoyments, are plagued with tormenting pains, and so forth—circumstances which certainly deserve mention in making a prognosis.

Treatment.

This is a difficult subject. The disturbance of nutrition in the cord is not so easily to be removed; and the patient is usually irritable, changeable, weak of purpose, so that it is often very hard to secure the necessary persistence and energy in treatment.

First of all, we must try to *remove the causes.* By reference to the list already given, it will appear what is implied by this.

In the *direct treatment,* the chief object doubtless consists in improvement of the nutrition and tone of the nervous system, especially the spinal cord. The first thing to prescribe is, therefore, in most cases, a *general tonic regimen:* good *food in abundance,* a not too sparing use of *spirituous drinks* (the English recommend them in large doses, and Hammond orders stimulants such as brandy and rum). As tonics, *quinia* and *iron,* preparations of zinc, and cod-liver oil may be used. A great deal of *fresh air* is indispensable to all the patients; active and passive *exercise* in the open air is therefore always indicated, but this should not be overdone, as frequent repose in the horizontal posture is often necessary. When it can be had, the air of *mountains and forests* is to be sought; a moderate *cold-water treatment* supports these tonic measures, and will be especially valuable when applied in a high mountain climate.

Strychnia (with other preparations of nux vomica) enjoys a special reputation with many physicians for the cure of this affection. It is given alone, or in suitable combination with other medicines. Thus, Hammond advises a combination of extract of nux vomica, half a grain (0.03), with phosphide of zinc, one-twelfth of a grain (0.005), given several times a day. A mixture of iron, quinia, and nux vomica in various forms seems often useful.

Another important remedy is the *galvanic current*. Hammond ascribes great successes to it, and I have also observed some good results. The best plan seems to consist in passing an ascending stabile current through the vertebral column, including the painful portions between the two poles. The current ought not to be very strong, and the duration of the applications must be short. The negative pole, acting directly on the painful vertebræ, has often been found to be of value. Many patients of this class will be benefited by the methods of general faradization and central galvanization. (See above, p. 181 et seq.)

Finally, *derivatives* have long enjoyed a general reputation. The best place for application seems to be directly upon the affected spots, the most painful parts of the spine. Many wonderful reports are made of the successful application of blisters, tartar-emetic ointment, oil of turpentine, veratrin ointment, etc. A repeated and continued use of these remedies is, however, often necessary. In less severe cases, dry cups suffice, and moxæ or the hot iron will be resorted to very rarely.

We should be careful about drawing blood from the spine, which was formerly so much in vogue. With most patients this does not agree; though in some cases, when the person is very robust and of full habit, or when there are signs of congestion, it may be very suitable.

A *symptomatic treatment* is required for the pains in the back and other neuralgiform symptoms. Hammond recommends opiates especially; the application of hot water or sand along the spine may also be tried; also bromide of potassium, blisters, and other derivatives, faradization, galvanization etc. For weakness, electricity is of special value.

8. *Functional Weakness of the Spinal Cord.—Spinal Nervous Weakness.—Neurasthenia Spinalis.*

Beard and *Rockwell*, Practical Treatise on the Uses of Electricity, etc. 1871. p. 294.—*Russel*, Cases of Paraplegia induced by Exhaustion of the Spinal Cord. Medical Times. Oct. 31, 1863; May 25, 1867.—*A. Bourbon*, De l'influence du coït et de l'onanisme dans la station sur la production de paraplégies. Paris, 1859.—*Leyden*, l. c. II. p. 22.—*Erb*, Bericht über die Versammlung mittelrhein. Aerzte am 18. Mai, 1875, in Heidelberg. Betz' Memorabil. 1875. 5. Heft.

Introduction and Definition.—Every physician encounters cases in the course of his daily practice, chiefly if not wholly originating in the higher walks of life, which may affect either one of the various departments of the nervous system. As a rule, there is no anatomical basis for them; and certainly, any severe anatomical changes seem to be excluded by the entire nature and course of the symptoms observed. Such are the cases which have been classed together under the name of "nervousness," "nervosismus," "nervous weakness," etc., and are commonly regarded with some mistrust by physicians. Beard and Rockwell have given a very good description of them, and have proposed the name *neurasthenia* (weakness of the nerves).

It is desirable to examine these cases more closely, and form them into classes. Careful observation quickly shows that this nervous weakness is capable of assuming various forms, and affecting different parts of the nervous system. In some cases the entire nervous system is more or less affected; in others the brain is chiefly affected, and in others still, the functions of the cord. It is this latter, the spinal form of neurasthenia, that we wish to speak of.

Abundant experience has shown me that these cases are not rare, and are of great practical consequence. For they cause much anxiety, not only to the patient, but also to the physician, owing to the striking resemblance they possess to the first stage of severe disease of the cord. It is of course important to decide this question as early as possible, as the prognosis of the two diseases must be very unlike.

We therefore understand by *spinal nervous weakness* those diseased conditions in which *marked and unquestionable disturbances of the functions of the cord exist, for which no considerable anatomical basis can be found or assumed;* a disease, therefore, which must at present be classed among the functional disorders.

Whether, and how often, this condition may lead to actual organic disease of the cord, cannot be known at present; from my experience I infer it to be rarely the case. The symptoms, however, are not infrequently present during the early period of anatomical lesions of the cord, though probably always associated with other disturbances, which enable us to recognize the commencement of the real disease.

It cannot be denied that this complaint has a close resemblance in many respects to spinal irritation, the subject of the previous section; and the opinion might perhaps be defended, that this disease is essentially, for the male sex, that which corresponds with spinal irritation in females. Nevertheless, characteristic differences will be seen as the description is given; I believe that the two diseases, though related, cannot be regarded as identical. It would be very desirable to lay out a better division and classification of these spinal "neuroses" by means of accurate clinical and symptomatic study, in order to promote the pathology of such an obscure subject.

What follows professes only to be a first step in this direction. Few accounts of the disease are recorded in print; I find an exquisite case briefly described in O. Berger;[1] Scholz[2] describes one under another name; that which Leyden describes as "spinal irritation from loss of semen" belongs for the most part to this class; so does much of what has been described as the results of spermatorrhœa, etc., in regard to which see Curschmann's excellent account in Vol. VIII. of this Cyclopædia.

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[1] Zur Pathogenese der Hemikranie. Virch. Arch. Bd. 59. p. 335. 1874.

[2] Ueber Rückenmarkslähmungen und deren Behandlung in Cudowa. p. 21.

Etiology.

A *predisposition* to this affection is most common in the male sex; and males are attacked in much larger numbers. Youth and middle age are also most exposed to the disease.

The chief contingent to this army of sufferers comes from neuropathic families, in which psychoses, hysteria, and other neuroses are well-known guests. The upper classes are also more affected than the lower, though the latter are by no means spared.

Finally, all the direct causes of the disease (to be named presently) may increase the predisposition, or perhaps cause it.

Among these *direct causes*, I am able from experience to name three as particularly active.

Excessive mental efforts may often produce the spinal form of neurasthenia—such as are made in the pursuit of a profession, or in severe mental toil, especially by night; a similar effect is produced by grief and excitement, violent excitement of the affections and passions, gaming, etc., in predisposed persons.

A much more frequent and important cause is found in *sexual excesses;* onanism begun very young and long continued; excessive coitus. I have often seen the entire series of symptoms develop in otherwise healthy men after very great sexual excesses, and again disappear in a few weeks under suitable observances. The standard of sexual "excess" is of course very varying; but, for the individuals in question, excess usually begins with comparatively moderate performances. Excesses of a less degree, but protracted, are often also at fault, and the affection not rarely appears after the honeymoon. In predisposed individuals, frequently repeated sexual excitement without gratification acts similarly; and the practice of having connection in the standing posture, mentioned by French authors, seems not less injurious.

Excessive bodily effort seems to be a less potent cause; yet long forced marches, mountain climbing, etc., are often mentioned as causes.

The injury is most distinct when several of the above causes coexist—*e. g.*, great mental and bodily overwork, with disturbance of the nightly rest (hence, the disease is not infrequent among physicians); or when, amid great mental exertions, sexual excesses are indulged in, etc.

It is not yet certain whether there are other causes; but it is probable that severe exhausting diseases, bad food, and other circumstances which depress the powers of the nervous system, may be active in this direction.

Symptoms.

The description of these is based almost exclusively upon the patients' subjective complaints. The persons affected are usually young or middle-aged men; they complain chiefly of a set of *motor disturbances*, principally consisting of a striking weakness and rapid fatigue of the lower extremities. They have constantly the sensation of great exhaustion of the legs, such as well persons only feel after making considerable exertions of body; this is perceived even in bed in the morning. They are incapable of walking or standing for a long time; a continuance in the standing posture is especially apt to make them tired. After more severe exertions, the great fatigue is accompanied by occasional tremor of the legs, and such a remarkable stiffness of the members as a well man would only feel after a very severe march. Unaccustomed efforts, even of a very moderate degree, are remarkably apt to produce that peculiar muscular pain, the origin of which is so obscure.

I refer to that well-known pain in the muscles which frequently occurs in well men after very active and unaccustomed muscular efforts, as when one rides, practises gymnastics, takes mountain walks, etc., after a long period of intermission. It usually appears about twenty-four hours after the exertion, is associated with slight swelling of the muscle and sensitiveness to pressure, and is produced by every contraction of the muscle affected. The essential nature of the pain is not yet known. Such a pain occurs in patients of the present class, after comparatively very slight exertions.

Similar symptoms of quick exhaustion and want of endurance are observed in the arms, but much less in degree than in the legs.

With this is associated a variety of *disturbances of the sen-*

sory organs. One of the most common is a peculiar *pain in the back*, seemingly localized in the muscles of that part, and appearing whenever certain movements are made, as of bending the spinal column forward or backward, or certain movements of the shoulder; and often in breathing or swallowing. The pain is not very intense, and varies greatly in its occurrence and position, rarely remaining for any length of time in one place. It is increased or brought on by slight exposure to cold, a draft of air, and probably also by excesses in Baccho aut Venere.

A diffused sensation of burning in the skin of the back is often observed, especially between the shoulder-blades; this is usually accompanied by sensitiveness of some of the spinous processes, just as in spinal irritation. In other cases, pain in the loins is observed.

In the extremities, especially the lower, this excessive feeling of fatigue often increases to that of slight *shooting and tearing pain* in the districts of certain nerves. These pains are usually not very severe, and pass away rapidly; they chiefly occur during movement and after unusual exertion. Some of the muscles, also, are not infrequently stiff and painful. In such cases there are seldom or never any defined and violent pains of the well-known lancinating sort (see above, p. 75-6).

Marked *paræsthesia* is also very rarely spoken of; many complain of a slight numbness or formication, especially in connection with great cold of the feet. A medical man, or one who knows and dreads the symptoms, is apt to speak of this.

Cold hands and feet are very commonly complained of; they are probably due, for the most part, to vaso-motor disturbances. The feet, in particular, are often icy cold, and can hardly be warmed, even in bed. The feeling of burning in the feet is rare, and if it occurs, is associated with an actual rise of local temperature.

The disturbances of the *sexual functions* are usually very striking; they commonly take the form of irritable weakness; the power of erection and of performing the act are lessened; the ejaculation takes place too quickly, and a repetition is impossible. The act is usually followed by remarkable prostration, restlessness of the limbs, and the like; or the man falls into a half-slumber and a profuse perspiration, etc. All the symptoms usually increase after the passion has been gratified, unless the greatest moderation has been used, or even after repeated sexual excitement. Pollutions or spermatorrhœa do not constitute a regular feature of the disease, unless they previously existed, and are to be considered as causes of the disease. Their occurrence, however, often aggravates the trouble, and they are much dreaded by the patient.

The *functions of the bladder* are usually quite normal; in some cases a little dribbling is spoken of. The sphincter ani acts properly. Trophic disturbances of the lower extremities, and bed-sores and the like, never occur.

With these regular and common symptoms we often find a number of others which indicate a wide extension of the nervous weakness. Among these we should mention *sleeplessness*, not usually very troublesome, but often peculiar in form. The patient wakes, after a few hours' sleep, with a sense of great uneasiness in his limbs, and cannot fall asleep for some hours after; in the morning he feels unusually prostrated. In some cases there is a certain sense of constriction of the head, timidity, often a remarkably womanish disposition, a tendency to weep, etc. I have seldom heard complaints of vertigo. The higher functions of the brain, memory, intelligence, etc., and the organs of special sense, remain perfectly normal. The power of mental work may be unimpaired, but is usually weakened by the patient's hypochondriacal depression.

The vegetative functions are unimpaired, on the whole; the disturbances most frequently observed are those of the digestive apparatus—dyspepsia, tendency to constipation, flatulence, etc. There is frequent complaint of palpitations, and a sense of constriction.

The *general sense of illness* is very great. Most patients are hypochondriacal in their feelings, are in dread of tabes, etc.; if a physician is the sufferer, he is apt to let his mind dwell on this anticipation, and to be made wretched by the thought.

The *general nutrition* is usually somewhat impaired; the patient loses some flesh, gets a sallow look, and becomes a little anæmic. There is always great sensitiveness to cold and severe changes of weather.

As compared with all these complaints, the *objective symptoms*—and this is a decisive point—are excessively slight, in fact, almost wholly negative. The closest examination shows no trace of disturbance of motility; all movements are performed easily and securely; the finest and most complicated actions can be executed; the patient stands on one foot, or with closed eyes, quite perfectly; only the power of endurance in muscular action is weakened.

In like manner there exists not the least disturbance of sensibility. There is usually no sensitiveness of the spinous processes. The reflex functions of the skin and tendons are usually normal. There is no atrophy, no change of electrical reaction. The only distinct symptom that can be found is, in many cases, a moderate degree of anæmia, a changed, suffering expression.

The closest objective study, therefore, discovers no change whatever that can stand in any relation to the patient's subjective complaints.

Of course, not all cases are alike, for many variations in the character of the disease occur; various symptoms may be absent, or may be more distinct in one case than in another; but the leading features of the disease will probably be traced in most cases.

I select from my list of recorded cases (of which I possess more than two dozen) the following one as an example: The patient, a wholesale merchant, aged thirty-five, belongs to a neuropathic family; two sisters were in the insane asylum; a brother has a tendency to melancholy and nervous complaints; he himself has been a long time "nervous." Married at twenty-three; he has three children; he says that he indulged a good deal in the sexual act, perhaps too much, but never observed any ill results from it. He has often been to bathe in the ocean, with temporary benefit. He has a great deal of work; at least eight hours a day in his office, occasionally going into a close, damp warehouse. All the nervous symptoms have slowly increased; they have been about as follows for four weeks past:

Great *general sense of fatigue*—this is very marked in the morning in bed; inability to walk for a long time, or, if he does it, it is followed by great fatigue and active *tremor of the legs*. When moderate exertion of an unwonted sort is made, there are *severe muscular pains* on the following day (as lately, after skating for fifteen minutes). No tottering or uncertainty of gait; no vertigo. Some sense of *fatigue in the arms*, uncertainty in writing.

No pains, no numbness or formication in legs or arms. No headache; only a frequent sense of pressure on the vertex. Intelligence and memory good; depressed, hypochondriacal state of feeling. Occasional *disagreeable feeling in the back*, but no real pain. Suffers much from *cold feet*, which formerly was never the case. Great *sensitiveness to cold;* after exposure he feels slight shooting pains in the limbs. *Sleeps badly;* usually wakes about three o'clock and remains awake for two or three hours, with great prostration and restlessness of limbs.

Vesical functions quite normal. *Sexual functions distinctly altered* in the last few weeks; sexual excitability increased, ejaculation too early, erection insufficient; after coitus a sense of exhaustion, with excitement and restless half-sleep.

Tendency to shed tears; unusual timidity and want of self-possession; noticeable confusion when he is conscious of being observed. Frequent palpitation, and some shortness of breath when he ascends stairs. Appetite and stools good.

Objective symptoms.—An apparently strong and well-nourished man; internal organs all sound. Motility quite normal by objective tests. Stands with closed eyes very well. No disturbance of sensibility. Cerebral nerves all normal. Slight anæmia.

He was ordered quinine and iron; cold friction in the morning; movement in the open air; strong diet; moderation in business and sexual act; afterwards a period of residence in a high mountain region.

Half a year later the patient returned from Switzerland and called upon me; he was considerably improved. The strength and endurance of the legs are decidedly greater, and his temper is much more cheerful. He walks his four or five hours every day and seldom suffers from tremor, still less from pain of the muscles. He has no cold feet, and his sensitiveness to cold is less. He sleeps much better, though not perfectly well. Sexual functions the least improved of all. Head never troubles him. Temper much improved; he has no disposition to weep.

After another half-year the greater part of the morbid symptoms had disappeared.

Course, duration, termination.—The disease usually begins in a very gradual and insidious way, yet sometimes it happens that it develops quite rapidly; some injury, a severe exertion, an excess, may bring the disease to an outbreak, when in the course of a few days or weeks it may reach a certain severity. In such acute cases there is during the first few days a marked and general sense of being ill, prostration, loss of appetite, etc.

The symptoms usually increase gradually for weeks and months, and then remain more or less stationary. Great fluctuations, considerable transitory improvements, are rare; but slight fluctuations in the intensity of the symptoms are common.

If a proper treatment and regimen are then adopted, gradual improvement occurs; but months and years may pass before the last traces are removed. Intercurrent febrile affections often seem to have a favorable influence upon the disease and to hasten recovery. Traces of the disease may last many years. Relapses are not uncommon, and are often occasioned by fresh injurious exposures of a trifling nature.

I am unable to state whether there are incurable cases, and whether the disease may last a great many years. At all events, cases occur of sufficient severity to compel the patient to give up business and avoid society, and to make his existence wretched.

I am also in doubt whether the disease can pass into any tangible chronic form of spinal disease (myelitis, sclerosis, gray degeneration). I have not observed such an event, and have never had to modify my diagnosis in the direction indicated. But only continued experience can decide.

Of the *essential nature* of the disease, I think we are not yet prepared to express an opinion. We are far too little acquainted with the pathology of many morbid processes of the cord, to allow ourselves to express a distinct opinion regarding the foundation of the disease here described.

A few remarks may, however, be allowed.

A spinal difficulty may naturally and reasonably be thought of, in connection with the symptoms described; the simultaneous occurrence of sensory and motor difficulties in both legs, the vaso-motor disturbances, the pains in the back, and especially the disturbances of the sexual function, which are most easily explained by increased excitability and weakness of the centres in the lumbar cord, are so strongly in favor of the spinal location of the disease, that other possibilities (as, for example, that of an affection of the cauda equina) sink into the shade by its side. The hypothesis of a spinal affection certainly seems to us the most acceptable.

Furthermore, the disease can only be a *functional disorder* in the ordinary sense of the term. In favor of this we have the absence of all objective disturbances, the absence of all paralytic symptoms, and the usually favorable course. In such circumstances it is hardly possible to suppose a considerable anatomical change in the nervous or interstitial structure of the cord;

the most would be, perhaps, a disturbance of circulation, hyperæmia, or anæmia of the cord. The regularly accepted description of these forms, however, does not agree with that of neurasthenia spinalis. I must admit that the hypothesis of anæmia of the cord is very plausible. But it is impossible at present to confirm this hypothesis in any way, and it must remain an open question.

It seems most natural to recur to *fine disturbances of nutrition* in the cord, such as we are still obliged to assume in so many diseases of the nervous system.

The term "irritable weakness" (reizbare Schwäche) agrees best with this notion, and in our description the weakness is quite prominent. We do not know at all what may be the nature of the processes of nutrition which cause the symptoms. We, however, believe that we are certainly entitled to locate them in the cord, especially the lower portion, the lumbar region. The most obvious view is that which supposes that the physiological fatigue of the nervous elements, which always occurs after severe and protracted irritation, becomes exaggerated and assumes a fixed form. In such a case we may suppose that the fatigue of the nervous elements does not become repaired in the prompt manner which is usual under physiological conditions.

Diagnosis.

The decision will be based chiefly on the great *disproportion between the acute subjective complaints of the patient and the almost negative result of objective examination.* The absence of each and every disturbance of motility and sensibility, of all symptoms which indicate an anatomical lesion of the cord, must give probability to the supposition of a purely functional disturbance. If in addition there exist general nervous weakness, sleeplessness, psychical irritability, a neuropathic constitution, and other etiological elements (especially that of undue sexual stimulation), the supposition then becomes much more probable. A good deal of practical experience and accuracy in examination is always necessary, and a long period of observation will often be necessary before we are clear in our minds.

A few remarks upon the points of diagnosis in reference to the better-known diseases of the cord will be useful here.

From a *commencing tabes*, which is the disease most usually suspected, spinal neurasthenia can be distinguished with tolerable ease.

The absence of lancinating pains, of paræsthesiæ and disturbances of sensibility, of the girdle-sensation, of tottering when the eyes are shut or in the dark, of motor uncertainty, of ataxia, will suffice. The study of the tendinous reflex action will perhaps furnish an important point, if it should appear that this reaction is absent in the early stages of tabes.

From *active hyperæmia of the* cord we may distinguish spinal neurasthenia by the absence of pain, of cutaneous hyperæsthesia, of symptoms of motor irritation, and probably also by the long duration of the disease. From *passive hyperæmia*, by the absence of paretic symptoms, of the feeling of heaviness in the legs, and probably also by the causes of the disease.

From *incipient myelitis* the diagnosis will be rendered possible by the absence of paræsthesiæ and anæsthesia, of paresis and paralysis, of weakness of the bladder, etc.

The distinction from *spinal irritation* will often be less easy to make. If we consider that the latter is more characterized by symptoms of sensory irritation, that in it the dorsal pains, neuralgias, sensitiveness of the vertebræ, etc., are in the foreground, while in spinal weakness the motor disability, the sexual weakness compose the chief subjects of complaint, we shall usually be in the right way. It must be admitted that there are cases of ambiguous signification which stand, as it were, half-way between the two forms of disease, and possess somewhat of each.

In no case can we state the diagnosis with certainty until after a most careful objective examination and a weighing of all circumstances, and, if possible, an opportunity for watching the case some time.

The *prognosis* is very essentially influenced by these circumstances. It is favorable, as compared with that of organic diseases of the cord, which somewhat resemble it in symptoms.

In most cases the patient recovers when the causes are removed and a suitable regimen of life is observed. Much time is required for this, and the patient must submit for months, or even years, to many a deprivation of ordinary enjoyments. If he will do this, his powers, and especially those of the mind, may remain considerable, provided that great regularity of life is observed, and all excesses are avoided.

In almost all cases, however, patients will have to suffer some loss of the former enjoyments of health; they remain a long time —perhaps always—in the category of "nervous" persons, and must look forward to a relapse of their disease with every fresh exposure to injurious influences.

If the hereditary tendency is very great, if there are unfavorable external conditions, permanent causes, the prognosis will of course be much less bright. The disease then continues, but without, as it seems, involving immediate danger to life. As regards the possibility of the development of anatomical lesions, we cannot at present render an opinion.

Treatment.

The *causal indication* must be satisfied, first and foremost. The excessive claims upon the nervous system must be decidedly forbidden, and in most cases it is really necessary to interpose a period of absolute rest from harmful occupations. The arrangements for this will differ in each case, according to the existing cause.

Especial attention is required by the *regimen and diet* of the patient. He must live a regular and healthy life in every respect, and must continue this plan with the greatest perseverance. He must work little, and only at fixed hours, with frequent interruptions; must go to bed early and sleep as much as he can; must have an abundance of strong, easily digestible food, at not too great intervals; spirituous drinks are allowable in moderation; much moving about in the open air (but never to the point of exhaustion, and with proper alternations; walks, mountain climbing, skating, gymnastics, etc.) is absolutely necessary; patients who are very easily exhausted must sit a great deal out of doors in a good air; the sexual act must be restricted as much as possible, but need not be absolutely forbidden in most cases, or but for a time; sexual excitement without gratification must be avoided as much as possible.

Of the plans of treatment for the direct relief of the disease, the use of a moderate cold-water cure, well graduated to the patient's strength and sensitiveness, deserves especial mention. Rubbing down with partly warmed water, gradually made colder, washing of the back, of the feet, and sitz-baths are the most suitable, and usually soon impart an increase of vigor and power. Douches and very cold applications should be avoided.

I have found the *mountain air* no less efficacious in many cases; a considerable time passed among high mountains, gradually going to higher stations, does such patients a great deal of good, and quickly improves the power of their lower extremities. It is well to combine this air-cure with a moderate water-cure, when possible. In selecting a place, its altitude, the quality of the service, and the convenience of the walks must be regarded; there is no lack of very suitable places in Switzerland and Tyrol.

The galvanic current has also an important place among the remedies for spinal nervous weakness; it is applied to the back in the usual way (best in the ascending direction, stabile, with change of the position of the electrodes—current *not too strong*), and may also be applied directly to the legs, and perhaps to the genitals also. Most patients bear it well, and are improved by it.

Of drugs, almost the only ones in use to be recommended are the preparations of *iron* and *quinia;* their forms and combinations may be very various. It is often useful to combine them, in the way recommended by Hammond (see the previous section), with small doses of nux vomica or strychnia; but it is well to be cautious. According to circumstances, other tonics may be used.

We shall often have to decide upon the choice and applicability of *baths.* For anæmic and debilitated persons, the *chalybeate baths* will be indicated; patients who require much protection, who are very sensitive to cold, should at first be sent to the *thermal brine baths* (Rehme, Nauheim, etc.), rather than to a cold-water cure. Sea-baths are a most excellent after-treatment for patients who are used to the water and have a good digestion.

Under all circumstances, these cures must be followed out steadily and repeatedly for a long time, for the complaint is very slow and does not yield to the first attack.

Symptomatic indications, such as arise from the presence of sleeplessness, pain, spermatorrhœa or pollutions, impotence, digestive disorders, etc., should be treated with the customary remedies.

9. *Inflammation of the Spinal Cord—Myelitis.*

Harless, Diss. inaug. de myelitide. Erlangen, 1814.—*Klohss*, De myelitide. Halae, 1820.—*Funk*, Die Rückenmarksentzündung. Bamberg, 1825.—*Albers*, Beob. auf d. Gebiete d. Pathol. I. S. 73. 1836.—*Ollivier*, l. c. II. S. 302. 3. Aufl.—*Abercrombie*, Krkh. d. Gehirns u. R.-M. Deutsch von G. v. d. Busch. 1829. S. 474.—*Cruveilhier*, Anatom. patholog. 1835. 42. Livr. 32 u. 38.—*Marcel*, Diagnost. et nature du ramolliss. blanc d. l. moelle épin. Gaz. méd. d. Paris. 1854. No. 52.—*Evan Reeves*, Acute Myelitis. Edinb. Med. Jour. I. 1855, 1856. p. 305 and 416.—*Oppolzer*, Acut. Entzünd. mit part. Erweichung d. R.-M. Spitalszeit. 1860. No. 1-3.—*Brown-Séquard*, Lect. on Diagn. and Treat. of the Prin. Forms of Paral. of the Lower Extrem. London, 1861.—*Koehler*, Monographie d. Meningit. Spinal. 1861. Beob. 17 u. 18.—*Mannkopf*, Fall von acut. Myelit Berl. klin. Woch. 1864. No. 1; u. Tagebl. d. 40. Vers. Deutsch. Aerzte u. Naturf. etc. 1865.—*Jaccoud*, Des paraplégies et de l'ataxie du mouv. Paris, 1864.—*Armin. Levy*, De myelit. spinali acuta. Diss. Berolin, 1863.—*Frommann*, Untersuch. über die normale u. patholog. Anatom. d. R.-M. I. 1864. II. 1867. —*Engelken*, Beitr. zur Pathol d. acuten Myelitis. Diss. Zürich, 1867.— *P. A. H. Sachse*, Ueber Myelit. etc. Diss. Berlin, 1867.—*Voisin*, Meningo-myélite aiguë. occas. p. l. froid. Gaz. d. hôp. 1865. Nos. 25–30.—*Harley* and *Lockhart Clarke*, Fatal Case of Acute Progress. Paral. from Softening etc. Lancet. 1868. Oct. 3.—*Keen*, Softening of the Spinal Cord, etc. Amer. Jour. of Med. Sci. July, 1869. p. 128.—*Hasse*, Krkht. des Nervensyst. 2. Aufl. S. 696.—*Hammond*, Diseases of the Nervous Syst. 3d edit. p. 456.—*M. Rosenthal*, Klinik d. Nervenkrkht. 2. Aufl. S. 296.—*Leyden*, Klinik d. Rückenmarkskrkht. II. S. 115.—*Dujardin-Beaumetz*, De la myélite aiguë. Paris, 1872.—*G. Hayem*, Des hémorrhag. intrarhachid. Paris, 1872.—*Hayem*, Deux cas de myél. aiguë centrale et diffuse. Arch. de Physiol. VI. p. 603 1874.—*C. Westphal*, Ueber fleckweise oder diseminirte Myelitis. Arch. f. Psych. u. Nerv. IV. S. 338. 1874.—*Bernheim*, Artikel: Myélite im Dict. Encyclop. des Sci. médic. II. sér. T. VIII. p. 674. 1874.—*Duchenne* (de Boulogne), Electris. localisée. 3e édit. 1872. p. 459.—*Hallopeau*, Études sur les myélites chron. diffuses. Arch. génér. d. méd. 6. sér. T. XVIII. u. XIX. 1871, 1872.—*C. Lange*, Fall von Mycl. interstit. chron. Hosp. Tid. 14. Aarg. S. 35. Virchow-Hirsch, Jahresb. pro 1871. II. S. 77.—*Frommann*, Fall von Wirbelcaries u. Degenerat. des R.-M. Virchow's Arch.

Bd. 54. 1872.—*Rob. W. Tibbits*, Case of Myelitis. Med. Times. 1871. May 13. —*Hallopeau*, Étude d. l. sclér. diff. periependymaire. Gaz. méd. 1870. Nos. 30–35.—*Vulpian*, Cas d. méning. spin. et d. sclérose corticale annulaire, etc. Arch. d. Physiol. II. p. 279. 1869.—*Martineau*, Inflammat. aiguë général. de l. substance grise d. l. moelle. Union méd. 1874. Nr. 30.—*Feinberg*, Ueber reflect. Gefässnervenlähmung u. Rückenmarksaffection nach Ueberfirnissen der Thiere. Virchow's Arch. Bd. 59. S. 270. 1874.—*Schueppel*, Fall von allg. Anästh. Arch. d. Heilk. XV. S. 44. 1874.—*Troisier*, Deux cas de lésions sclér. de la moëlle ép. Arch. d. Physiol. V. p. 709. 1873.—*Joffroy*, Faits experim. pour servir à l'histoire de la myélite. Gaz. méd. de Paris. 1873. Nr. 36.—*Charcot*, Sur la tuméfact. des cellules nerv. motrices et des cylindres axiles, etc. Arch. d. Physiol. IV. p. 93. 1872.—*C. Lange*, Bidrag til etc. Hosp. Tid. 16. Aarg., Schmidt's Jahrbb. Bd. 168. S. 238.—*Raymond*, Myél. de la région dorsale. Gaz. méd. 1874. No. 9.—*F. Raymond*, Myélite de la rég. cervicale. Progrès méd. 1875. No. 17.—*Gérin-Rose*, Myél. aiguë généralisée. Union méd. 1875. No. 90.—*Langhans*, Myelitis d. grauen Commiss. etc. bei Lepra anæsth. Virch. Arch. Bd. 64. 1875.—*Hamilton*, On Myelitis, etc. Quart. Journ. of Microsc. Sci. Oct. 1875. p. 334.—*Pierret*, Note sur un cas de myél. à rechutes. Arch. d. Physiol. VIII. p. 45. 1876.—*Laveran*, Obs. d. myél. centr. subaiguë. Ibid. VII. p. 866. 1875.—*P. Baumgarten*, Eigenth. Fall von Paralys. ascend. acut. mit Pilzbildung im Blut. Arch. d. Heilk. XVII. S. 245. 1876.—*Lewin*, Paraplegie in Folge von acut. Myelitis u. el. Behandlung derselben. Deutsche Klinik. 1875. Nr. 11.—See also the literature of multiple sclerosis, sclerosis of the posterior columns, lateral sclerosis, poliomyelitis anter. acuta et chronica, etc., in the succeeding chapters of this work.

Introduction.

Myelitis constitutes unquestionably the most extensive and important chapter in the pathology of the medulla spinalis. Any one who possesses even a superficial knowledge of the subject, will be able to appreciate the difficulties which confront us in the attempt to furnish a comprehensive and classical account of it.

The material at hand bearing on inflammation of the spinal cord and affections that are classed with it, is immense and in the critical sifting and arrangement of this material we meet with difficulties that cannot be overcome. These are encountered chiefly in the domain of pathological anatomy, many and perhaps most of the questions which come within the limits of this department being still in process of development. It is as yet impossible to define accurately the boundaries of inflammation of the spinal cord in both its acute and chronic forms, and to decide exactly what does and what does not fall within them. Some authors do not hesitate to class almost all the forms of acute as well as chronic disease of the cord, which will be described in the following chapters, under the head of Inflammation. They regard tabes dorsalis, the so-called lateral sclerosis, multiple sclerosis, progressive muscular atrophy, spinal paralysis of children, myelitis centralis, chronic atrophy of the cord, and secondary degeneration of it, as mere subvarieties or different localizations of one and the same inflammatory process. Others, on the contrary, hold that a number of these processes, such as simple and degenerative atrophy, softening, sclerosis, gray degeneration, and the like, are entirely distinct from and independent of inflammation.

Hence, the additions made by one party to the great mass of material bearing on the disease, are detached and diverted in different directions by others. It is as yet impossible to foretell when the elucidation of the facts and opinions will take place. Of late, however, all the anatomical and clinical investigations have aimed at this goal, and they have already thrown much light on some of the doubtful points.

We need, above all things, searching histological studies of the diseased spinal cord, to enable us to comprehend the genesis and the histological development of the different processes. There is, moreover, great need of further elucidation and a more exact definition of the general pathological conception of inflammation, in order to show what should be comprised under the head of inflammation of such an organ as the spinal cord. It is especially necessary that we should learn how chronic inflammation of the cord is to be defined, and how it is to be distinguished from, or in what genetic connection it stands to, the other forms of chronic disease, viz., atrophy, degeneration, softening, sclerosis, etc. Not till these steps have been gained, will it be possible to determine positively what forms of disease are to be classed under the head of myelitis, and to describe accurately the clinical histories of the different forms.

The necessities of practice, however, demand a dogmatic presentation of the subject, and the practical physician has a right to insist, that it should be in accord with the present state of our knowledge, however incomplete that may be, all disputed questions being avoided as far as possible. This idea has guided and influenced us in the preparation of the following account of the disease. It is both short and incomplete, but in extenuation of the latter we must plead the difficulty of the subject, and of the former the shortness of the space allotted us.

History.

An accurate history of the growth of medical knowledge on the subject of myelitis would be almost the same thing as a history of the pathology of the spinal cord in general. That does not enter into our present task. Nor is it at all necessary for us to investigate the question whether the ancients—Hippocrates, Aretæus, Galen, and others—had or had not any definite conception of myelitis. There can be no doubt that they repeatedly met with the disease, but they did not differentiate it sharply from other affections. We find in their works more or less recognizable descriptions of both the acute and the chronic forms of myelitis.

Our more accurate knowledge of the inflammations of the spinal cord dates only from the end of the last and the beginning of the present century. Here, too, it was P. Frank (1792) who gave the impetus to more accurate investigations. Spinitis, rachialgitis, and the like, were the terms then applied to the more acute forms of the disease. At a later period, the term "myelitis," which is now universally employed to designate inflammation of the spinal cord, was introduced, mainly through the works of Harless (1814) and Klohss (1820).

In the third decennium of this century, the epoch-making works of Ollivier (1st Edit., 1821) and of Abercrombie (1828) appeared and placed the subject of myelitis on a firmer foundation. These authors were the first to point out the intimate connection of softening with the acute form of the disease. After this the subject remained in statu quo for a long time. During the fifth and sixth decennia the attention of physicians was gradually directed more to the chronic forms of the disease. Among the valuable contributions of this period, we must mention Tuerck's discoveries concerning the secondary degenerations, and Romberg's account of tabes dorsalis, but, above all, the admirable observations and investigations of Duchenne (of Boulogne). This last-named author described in a classical manner the clinical histories of chronic spinal paralyses, locomotor ataxia, progressive muscular atrophy, etc.

The development of the pathology of the spinal cord did not, however, receive any great impetus until the beginning of the seventh decennary, when it began to make rapid progress under the influence of improved methods of physiological and histological investigation. In this accelerated development the subject of myelitis played a prominent part. We must here extol especially the works of Brown-Séquard. The publications of Oppolzer, Levy, Mannkopf, and others, on acute myelitis, are also deserving of special mention, while the pathological anatomy of the subacute and chronic forms of myelitis was cleared up very greatly by the valuable contributions of Frommann (1864 and 1867). From this time forward we have to chronicle a rapid advance in the knowledge of the acute and chronic forms of inflammation of the spinal cord, which became the object of zealous study in France and England, as well as in Germany. Broad foundations, on which to build up the edifice of acute myelitis, were laid by the works of Engelken, Charcot, Dujardin-Beaumetz, Hayem, Westphal, J. v. Heine, and others, but it was nevertheless the chronic forms of the disease which attracted the most earnest attention of scientific men. Our knowledge of these has been greatly advanced by the admirable works of Friedreich, Westphal, Leyden, Frommann, Th. Simon, and others, in Germany; of Lockhart Clarke, Gull, and others, in England; and of Duchenne, Topinard, Jaccoud, Charcot, Vulpian, Hallopeau, Joffroy, Michaud, Pierret, and others, in France.

The school of the Salpêtrière, under the direction of Charcot and Vulpian, has unquestionably played the most prominent part in the furtherance of this development. We have to thank it for numerous admirable works, full of new discoveries and fruitful thoughts. Germany, too, has furnished during recent years a number of excellent works. (Compare the histories of tabes dorsalis, multiple sclerosis, and spinal paralysis of children.) Finally, we must also mention the claims of the electro-therapeutists, especially those of Germany, whom we must thank for many valuable advances, not only in the treatment, but also in the pathology of myelitis.

In describing myelitis, it seems to us for many reasons expedient to distinguish two principal forms of the disease, viz., the *acute* and the *chronic*. These two forms run into one another in many points, but as a general thing they present noticeable differences, chiefly in regard to their course, but also in regard to their pathological anatomy and their clinical manifestations. The different forms and subvarieties of the disease, of which there are not a few, will be only briefly alluded to here, as we shall return more fully to them at a later period.

A. Acute Inflammation of the Spinal Cord—Myelitis Acuta.

Definition.—We understand by acute myelitis all the varieties of *acute inflammation of the substance of the spinal cord, which rapidly lead to serious disturbances, and are usually, if not always, attended by fever.*

We have to deal here with rapidly developing inflammatory changes, which affect the connective substance as well as the nervous elements of the cord, but of which all the histological steps are not yet thoroughly understood. The usual result is a rapid destruction of the histological constitution of the cord, which almost always takes the form of *softening*, and is of course accompanied by abolition of the function of the affected part. In the present state of our knowledge, it is scarcely possible to make an accurate subdivision of the disease into parenchymatous and interstitial acute myelitis.

Acute myelitis may present itself in different forms, according to its exact seat. It may also follow very different courses. It is true that the development of the disease and of the disturbances of function caused by it, is in almost all cases very rapid, but of the cases which prove fatal, it is only in a small number that the subsequent course of the disease is as rapid as its commencement. On the other hand, a speedy recovery is very rare; usually the disease runs a more or less protracted course, and either terminates in death or sinks into a chronic affection.

Etiology and Pathogenesis.

The comparative rarity of acute myelitis accounts in a measure for our lack of knowledge concerning its causes. It is only in a small number of the cases that we can succeed in discovering a tangible cause, and even in them its mode of action usually remains enveloped in mystery. Many cases apparently originate spontaneously, without any exciting cause at all. In such cases the cord has been subjected to the action of irritants which are entirely unknown to us.

Very little is known concerning the conditions which produce an increased *predisposition* to acute myelitis. The male sex seems to be more subject to the disease than the female. It is said that the greatest number of cases occur during youth and middle age, between the ages of ten and thirty years, but still there is a particular form of acute myelitis, the so-called spinal paralysis of children, which is essentially and almost exclusively a disease of childhood. Many authors hold that this form of the affection is connected with dentition, but the correctness of the opinion is questionable.

It seems to us that it would be more correct to class sexual excesses, the practice of coitus in the erect position, severe bodily exertion and the like, with the predisposing than with the exciting causes of acute myelitis.

Of the latter the simplest and the easiest to study are *injuries*, to which the spinal cord is so frequently exposed, and which may result from the most various external agencies. All sorts of injuries of the cord, such as those produced by puncturing and cutting instruments, by fractures and luxations of the vertebræ, contusions, etc., form the regular starting-point for the acute traumatic myelitis which we have already described in

No. 4, Part 1, p. 305. This traumatic myelitis has also repeatedly been the subject of experimental investigations.

Next in order come the cases, in which a slow *compression* of the spinal cord is the starting-point of a myelitis. This compression may be due to lesions of the most various sorts. (Comp. above, No. 5, Part 1, p. 319.) It is true that some of the compressing agents which come in question here, are more liable to excite the subacute and chronic forms of myelitis. When, however, the agent which produces compression of the cord, is at the same time of an irritating nature, *e. g.*, carcinoma, the inflammatory exudation in caries of the vertebræ, etc., changes are not infrequently produced, which unquestionably belong to acute myelitis. This brings us to those forms of acute myelitis which are due to the *transmission of inflammatory processes* from neighboring organs and tissues, without the intervention of compression of the cord. The principal, and practically the only important, disease belonging in this category is acute meningitis spinalis, which is almost invariably attended by more or less extensive implication of the cord itself. We have already spoken of this peculiarity in the chapter on Leptomeningitis Acuta, Part 1, p. 233.

Catching cold is unquestionably one of the most important and most frequent causes of acute myelitis. The cases in which exposure to some one of the more severe causes of colds, such as sudden cooling of the overheated body, a fall into water, sleeping on the damp earth or in snow, etc., has been followed immediately by the symptoms of acute myelitis, are too numerous to leave any doubt of the potentiality of this cause. (See the observations of Ollivier, Oppolzer, Voisin, and others.) Dujardin-Beaumetz believes that the frequency of the disease among bakers can be ascribed to the fact, that their work exposes them in an unusual degree to colds.

The question whether *excessive bodily exertion* must also be classed among the exciting causes, cannot as yet be decided. Perhaps it acts only as a predisposing cause. At all events, it seems to have a certain etiological significance, when combined with exposure to the causes of colds; every war, and especially every winter campaign, furnishes examples which illustrate this

The same may also be said of the different varieties of *sexual excess*.

Acute myelitis is not unfrequently developed as a complication or sequel of *acute diseases*. It has been observed in connection with typhus, the acute exanthemata, acute rheumatism, severe puerperal diseases, etc.; most frequently, however, in connection with variola (Westphal). In this last case the myelitis has set in at a variable period after the outbreak of the primary disease, in a few rare cases not until several weeks afterwards. Baumgarten recently reported a remarkable case, in which infection with bacteria (from malignant pustule) was in all probability the cause of an exceedingly acute myelitis.

Among the chronic infectious diseases, *syphilis* can unquestionably cause acute myelitis. A question may be raised as to whether we have to deal in this case with a specific luetic process, or with an ordinary non-specific myelitis of which the syphilis has been only a predisposing and distant cause. It is at all events certain, that myelitis running a rapid course, is observed with unusual frequency among syphilitic patients. Our own observations lead us to agree with Hayem, who claims that syphilis is an etiological agent of decided importance.

Continued suppression of the menses, sweating of the feet, and hemorrhoidal bleeding play a more or less doubtful rôle in the etiology of acute myelitis.

Violent *emotions* seem in many cases to have been really the starting-point of myelitis. At least some isolated cases have been reported (by Leyden, among others), in which the first symptoms of myelitis showed themselves immediately after some intense psychical movement, such as fear, anxiety, or anger.

Finally, *irritations and diseases of peripheral organs* must also be mentioned among the causes of acute myelitis, although the cases in which they are really the agens morbi are comparatively rare. For instance, a certain proportion of the so-called reflex paraplegias, which are developed in connection with diseases of the urinary and digestive apparatuses and of the uterus, or with irritations and inflammations of the peripheral nerves, the joints, etc., should really be classed as acute myelitis. This fact and the pathogenesis of reflex paralyses have already been discussed in more than one chapter of this work, and we may refer the inquirer to them for further information.[1] Here, we will only mention the fact that Feinberg[2] has recently succeeded, by varnishing the skin of rabbits, in exciting an acute affection of the spinal cord, which at all events closely resembles inflammation. He regards it as reflex in nature and secondary to the severe irritation of peripheral sensitive nerves, and ascribes it to paralysis of the vaso-motor centres. After all has been said, however, we must admit that we have very little positive knowledge concerning the pathogenesis of reflex paraplegias, and unfortunately our knowledge of the pathogenesis of myelitis due to other causes is equally deficient. The traumatic cases and those secondary forms due to extension of inflammation from neighboring tissues, are the easiest to comprehend. When, however, we seek to explain the manner in which catching cold or emotional disturbance produces myelitis, or the mode in which it is developed in connection with acute diseases, or with syphilis, etc., we can only bring forward more or less gratuitous theories, which it would be useless to discuss here.

Pathological Anatomy.

Frequently a spinal cord which is the seat of acute inflammation, presents very little or even absolutely no macroscopic change. There is not the slightest reason to doubt, that the disease has on innumerable occasions been overlooked at the autopsy. In fact, we can scarcely ever be absolutely certain that an acute myelitis exists, until a microscopic examination has been made.

In the macroscopic examination the most striking peculiarity is the diminution in the consistence, the *softening*, of the cord (myelomalacie). This change is so constant, that softening has been generally, but very incorrectly, identified with acute inflammation. That the two processes are not identical, is evident on the one hand from the fact, that every softened cord is not inflamed, and on the other from the fact that inflammation of the cord does not always terminate in softening. It would be better consequently to discard entirely the use of the term "softening," to designate inflammatory processes. I must at all events protest most earnestly against the promiscuous employment of the name myelomalacia as a synonym for myelitis. That term should be reserved for processes of softening pure and simple.

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[1] See Vol. XI. and Vol. XII.

[2] Virchow's Archiv. Vol. 59. p. 270.

The inflammatory changes, which we will consider first, vary greatly as regards their *localization* in the spinal cord. Acute myelitis has its seat and starting-point most frequently in the gray substance, a fact which depends probably on the vascular richness of this portion of the cord. It may extend to a greater or less distance in a vertical direction, and may penetrate more or less deeply into the white columns. This is the form which, since the time of Albers, has been usually designated as *myelitis centralis.*

It may spread very rapidly through the gray substance, and in a short time involve the greater part, or even the whole of the gray axis—*diffuse central myelitis* (Hayem). When the white columns are also involved to a marked extent, so that the entire cord is more or less implicated in the inflammatory process, the affection may be called *myelitis diffusa.* Strictly speaking, however, this is a rare occurrence.

Not unfrequently the entire thickness of the cord is affected for a longer or shorter distance. The diseased part may in such cases be several inches in length, but the inflammatory process is usually not equally intense throughout its whole extent. This is the form which is now ordinarily described as *myelitis transversa.*

When the centre of inflammation is less extensive, so that it involves only a small portion of the cord both vertically and transversely, it is termed *myelitis circumscripta.* Here we have to deal with a small spot of inflammation buried somewhere in the substance of the cord. Not unfrequently, however, we meet with several of these small spots in the same cord. Cases have been observed (Westphal), in which the myelitis presented itself in the form of numerous foci scattered throughout the entire cord and separated from one another by healthy tissue—cases in which the disease was widespread, but attacked only circumscribed and scattered spots. This is called *myelitis disseminata.*

Finally, that form of the disease which is secondary to meningitis, affects principally the peripheral layers of the cord, penetrating more or less deeply and extending vertically to a variable distance—*myelitis peripherica.*

The appearances presented by the affected spots vary greatly according to the stage of the myelitis, and in part also according to the special peculiarities of the process in the individual cases—always provided there are macroscopically demonstrable lesions. We must here distinguish different stages.

1. *The stage of hyperæmia and commencing exudation* (red softening). We do not often meet with this stage of the disease at the post-mortem table. The opportunities to study it are furnished most frequently by those cases in which the inflammation is secondary to severe traumatic lesions, or by cases of myelitis centralis, which run a rapidly fatal course.

At the affected spot a slight, and in rare cases a marked, swelling can be recognized. On section, the transverse markings are found to be blurred and indistinct, and the cut surface not unfrequently presents a variegated, marbled appearance. The red color of the tissue, which is due to the hyperæmia, is usually very marked; it may vary from a slight rosy injection to a deep red, reddish brown or chocolate color, when more or less numerous capillary hemorrhages are added to the often very intense hyperæmia. The inflamed spots are almost always unusually moist and soft; they swell up above the level of the cut surface. The softening may be so great that the tissue is reduced to the consistency of pap. In very rare cases we observe a slight increase in consistency at this stage, probably on account of infiltration of the interstitial tissue with a firmer exudation. Hayem has reported an instance of this. In some few cases we can see with the naked eye, or with the help of a lens, a distinct deposit of exudation in the affected spot; it takes the form of whitish or yellowish, clouded or glassy, colloid streaks or rings, which are imbedded in the septula or poured out around the vessels. The meninges in the neighborhood of the affected spot also frequently present the signs of hyperæmia and inflammation.

In preparations hardened in chromic acid, the myelitic spots can usually be much more readily distinguished from the healthy tissue by their different (brighter yellow) color, than in the fresh cord. The inflamed parts, moreover, harden slowly and badly, and on section they appear friable, crumbly and not coherent, and do not present sharply defined markings. The smaller spots of myelitis in particular, as well as the secondary degenerations, are very sharply and distinctly mapped out by this discoloration.

2. *The stage of fatty degeneration and of resorption* (yellow and white softening). As the disease progresses, the affected spot becomes constantly paler and more and more softened. Its color changes gradually from red to yellow; this change is due partly to the diffusion and alteration of the coloring matter of the blood, partly to the fatty degeneration of the medullary sheaths and the formation of masses of fat-granules, and partly to the cessation of the hyperæmia. Through the influence of these three agencies, but particularly in consequence of the accumulation of fatty detritus, the color becomes progressively whiter, and the entire substance assumes a creamy or milky appearance. The softening has meanwhile made rapid progress, the medulla swells up very much above the cut surface, acquires a pappy or even a more fluid consistency, and often flows out of the sac of the pia entirely. This softening is caused principally by the fluid exudation from the vessels, acting in concert with the destruction of the nerve-fibres. This exudation may possibly present certain differences in different cases, which would account for the varying degree of the softening, or even for its entire absence. Of this, however, we have no positive knowledge.

At last nothing remains of the diseased spot but the vascular network and a portion of the hypertrophied septa, between which is a softened mass that can readily be pressed out, or which perhaps flows out without the help of pressure. In consequence of the progressive resorption of the nerve-substance and the fat-granules, the spot gradually assumes a more grayish color, and finally becomes smaller and depressed.

Actual suppuration occurs very rarely in acute myelitis. When abscess of the cord does form, it is generally secondary to a severe traumatic lesion or to suppurative meningitis. In spontaneous myelitis, on the other hand, suppuration is exceedingly rare, and has only been observed in a very few cases.

3. *The terminal stage* (formation of cicatrices or cysts, induration, sclerosis, etc.).

The resorption of the softened masses continues until finally all the fluid portions have completely disappeared. Nothing remains but the vascular and connective tissue networks, which are in part thickened and hypertrophied; they form a more or less dense, shrivelled, grayish, semi-transparent cicatrix, which is often flecked with pigment. This cicatrix unites the portions of the cord that have remained healthy. In some rare cases a restoration of the nerve-tissue takes place; after a certain length of time small, dark-bordered, regenerated nerve-fibres can be demonstrated in the cicatrix. In this way also a restoration of function can be brought about, as happened in one of Charcot's cases. This fortunate result seems, however, to occur more readily in the subacute and chronic forms.

Not unfrequently more or less extensive collections of fluid are left behind in the framework of the cicatrix, and lead to the formation of single or multiple, large or small cysts. They usually contain a muddy fluid resembling milk, or more frequently, serum.

In many cases, on the contrary, the interstitial, supporting framework becomes in course of time greatly hypertrophied and consolidated. It increases in thickness and density, the vessels become larger and their walls thicker; the previously softened spot becomes firmer and denser, gray and semi-transparent, and presents a marked contrast in color and consistency to the surrounding parts. Here we have the commencement of *hardening*, or *sclerosis*. When the interstitial growth of connective tissue persists, and extends in a slow but progressive manner to the neighboring parts of the cord, which were previously either healthy or very slightly changed, and when it propagates itself there, leading to a gradual destruction of the nervous elements, we have before us *the transition into chronic myelitis.* This is a frequent occurrence. In almost all cases of myelitis which do not prove rapidly fatal, this transition takes place at some late period of the disease.

The macroscopic examination, as a rule, reveals also an *implication of the meninges* in the inflammatory process. The signs of it are cloudiness of the membranes, infiltration with serum, cellular elements or pus (often only discovered at the microscopical examination), thickening, adhesions, capillary extravasations in addition to extreme hyperæmia, etc. The *spinal fluid* is usually increased, somewhat cloudy and reddish, but it is sometimes entirely unchanged. The condition of the roots of the nerves is regulated by that of the pia; they are sometimes softened, loosened and swollen, sometimes atrophic, gray, translucent and indurated.

In the later stages, in addition to the actual centre of inflammation, we often meet with the so-called *secondary degenerations*, of which we have already repeatedly spoken: ascending degeneration in the funiculi graciles, and descending in the posterior lateral columns, etc. In many cases of acute myelitis the disease does not last long enough for their development, but they are rarely wanting in the cases that become chronic. The transition from myelitis to secondary degenerations can often be traced very accurately in successive transverse sections.

The *microscopical examination* is, however, of supreme importance for the understanding of the pathological processes which take place here. The cord should be examined microscopically, both in the fresh state and after it has been hardened by the usual methods. The changes are seen to affect the nerve-fibres and the ganglion-cells, as well as the connective tissue framework and its vessels.

In the *first stage* we find marked dilatation of the capillaries and small arteries, and more especially of the small veins; they are distended with blood, and not unfrequently enveloped in layers of white and red blood corpuscles arranged in the form of a sheath. Here and there larger collections of red corpuscles are found (capillary extravasations). The walls of the vessels are thickened and studded with fat-granules and granule-cells, presenting exudative and plastic infiltration. Hayem describes particularly a glassy, colloid exudation, which surrounds the vessels like a sheath in a layer of varying thickness, and which is also found disseminated elsewhere in the tissue.

Baumgarten seems to have seen something very similar—a hyaline, firm, glassy exudation in the tissue of the gray substance, about the vessels and in the anterior longitudinal furrow—in his interesting case of acute central myelitis, which was probably due to infection with the poison of anthrax.

Marked changes are always found in the *neuroglia*. The fibres of the reticulum are thickened and swollen, the network is much denser and more distinct, and it is in part filled with nuclei and cells. The *glia cells* themselves are swollen and increased in number; they often contain several nuclei, and are sometimes, like the thickened connective tissue septa, infiltrated with colloid masses. Even in this stage we generally find granule-cells in greater or less numbers, partly in the immediate neighborhood of the vessels, partly scattered irregularly in the interstitial tissue and its meshes.

The changes in the *nerve-fibres* are also very striking and important. The fibres, in general, present irregular contractions and enlargements; the medullary sheath has in places broken down into globules or larger irregular fragments, and has become granular; in many fibres it has entirely disappeared, or has united with the axis-cylinder to form a single, homogeneous mass. The *axis-cylinders* themselves, as is evident from the observations of Frommann, Charcot, Hayem, Joffroy and others, are frequently swollen, and often attain enormous dimensions. They present large, spindle-shaped swellings and bulging, club-shaped fragments; they are streaky, clouded, studded with granules, brittle, and apparently in a state of granular degeneration. The thickening of the axis-cylinder can be very readily seen in transverse sections, in which they are often found lying together in groups. Isolated swollen axis-cylinders are also found lying in the midst of unchanged nerve-fibres.

The *ganglion-cells* are also involved in the inflammatory changes. They are swollen, often to a very considerable size, looking then as if they were puffed up. Their substance is clouded; in the beginning the nucleus and nucleolus are usually intact, but in a few instances they are in process of division. Sometimes we observe glassy swelling of the cells, and a development of vacuoli in them. Their processes are also swollen, clouded, irregular in shape, and in part destroyed. At a late period the cells disappear before the advancing processes of destruction.

In the *second stage* we find the *nervous elements* fully involved in the process of destruction: the nerve-fibres are broken down into fragments; the medullary sheath is in a state of fatty degeneration, and the axis-cylinder altered or destroyed. The ganglion-cells are partly melted down and destroyed, partly atrophied and reduced to small glistening scales, without demonstrable structure. The *connective tissue framework* is loosened and softened by the fluid exudation, the development of cells and fatty degeneration. The *walls of the vessels* are thickened, very rich in nuclei, filled with fat-granules and frequently surrounded by thick layers of granular cells. The semi-fluid pulpy mass formed by the inflamed and softened cord, contains also numerous lumps and drops of myelin, the remains of blood-corpuscles, colorless cells, pigment-granules, fine granular detritus, and, above all, numerous fat-granule-cells. These *fat-granule-cells* form usually the most striking constituent of the softened myelitic mass, and attract the attention of the observer at once; they are especially distinct when the cord is examined in the fresh state. They are found deposited about the vessels, and in the spaces left empty by the destruction of the nerve-fibres and axis cylinders, and also in the septa of the neuroglia. This extensive distribution of these cells indicates of itself that they are produced in different ways; it is, in fact, probable that some of them are formed from young cellular elements which have taken up fat, while others can be ascribed to fatty degeneration of the glia cells, and still others to fatty destruction of the nerve-fibres and the spindle-shaped swellings of the axis-cylinders (Hayem, Th. Simon).

In the *third stage* we find in the affected spot, which is now engaged in an effort at cicatrization, a sclerotic connective tissue with numerous nuclei and neuroglia cells. At this period Deiter's cells often become very distinct and prominent; they are often large and possess numerous processes. The vessels are dilated, and their walls thickened. The ganglion-cells are mostly atrophied. Sometimes numerous small, delicate nerve-fibres, with thin medullary sheaths, can be seen.

Occasionally cysts are found, surrounded by a more or less dense layer of connective tissue, and usually also traversed by a large-meshed, connective tissue network. Their contents are partly muddy, partly serous; microscopically, nothing can as a rule be discovered in the fluid, except detritus.

In the stage of transition to sclerosis and chronic myelitis, the increase of the interstitial tissue becomes particularly distinct. The threads of the reticulum become wider and thicker, the spider-cells are enlarged and increased in number, the vessels are thickened, the nerve-fibres and ganglion-cells are involved in degenerative atrophy. The tissue contains relatively few fat-granule-cells, but, on the other hand, it contains large quantities of corpora amylacea.

When *secondary degeneration* exists, it presents the usual well-known histological characters, which will be described in detail in a subsequent chapter (No. 19).

A priori, the usefulness of *experimental investigations* of these conditions would seem self-evident. Unfortunately, however, the attempts to clear up the subject in this way have been but few in number, and have furnished no important, or at all events no conclusive results.

Strange to say, the numberless experimental sections of the spinal cord performed by physiologists, although undoubtedly on every occasion followed by traumatic myelitis, have not hitherto been utilized to any extent worth mentioning, for the study of this affection.

Dujardin-Beaumetz describes some experiments performed by Hayem and Liouville, who sought to excite myelitis by applying iodine or glycerine to the outside of the cord, or by injecting them into its substance; also some experiments undertaken by himself in connection with Grancher, in which mechanical injuries were employed to excite the inflammation. The results of these experiments are, however, very unsatisfactory. Joffroy made some similar experiments on dogs, and obtained the same histological changes as those which have been here described as belonging to the acute myelitis of men.

The investigations of Feinberg and also those of Klemm, who endeavored to produce myelitis in a reflex manner or by means of a neuritis migrans, have not made any important addition to our knowledge of the histological genesis of the myelitic changes.

A somewhat more comprehensive series of experiments was set on foot by Leyden, but his account of the results obtained, which was published in his "Klinik der Rückenmarkskrankheiten," is only fragmentary. He experimented on cats and dogs, and employed exclusively injections of Fowler's solution into the cord, the result being almost always a *suppurative* myelitis. As, however, this variety of the disease is exceedingly rare in man, the experiments are not of much value for the elucidation of the subject of acute myelitis in the human subject. Moreover, the reports that have thus far been published by Leyden, contain no conclusive information concerning the manner in which the process is developed, the relation of the minute histological changes to each other in point of time, and the part taken by the different tissue-elements in the process. Still, we have learned many things from these experiments. They have proved, for instance, that an injury of the sort here mentioned can produce an intense myelitis, which may extend beyond the point of irritation; that the process presents varying degrees of intensity in one and the same experiment; that the affection diminishes in intensity as it recedes from the point of irritation; that this diminution in intensity is indicated on the one hand, by a tendency to localization in scattered, circumscribed spots, and on the other, by a preponderance of the connective-tissue growth over the softening, liquefaction and suppuration; that multiplication and swelling of the nuclei of the neuroglia, swelling of the nerve-fibres and axis-cylinders, disappearance of the medullary sheath and development of granule-cells, must be regarded as the first signs of a commencing acute myelitis, etc.

In the latest experiments of Hamilton on cats, only the first days of acute traumatic myelitis are taken into consideration. He found among other things, that rounded bodies separate from the swollen axis-cylinders and either degenerate into colloid bodies, or give rise to an endogenous brood of young round cells (pus-cells). He also observed swelling and cloudiness of the ganglion-cells, multiplication of the nuclei of the neuroglia, collections of cells in the walls of the vessels, etc.

It is certainly desirable and even necessary that these experiments should be repeated with modifications, that myelitis should be produced in other ways, so as to bring the results more in accord with the lesions in man, and to furnish a more complete periscope of the process in its different modifications and its various degrees of intensity.

We must here mention briefly some special forms of acute myelitis. First of all, we will speak of *myelitis centralis acuta.* In this form the softening and liquefaction affects pre-eminently the gray substance, which flows out when the cord is divided, leaving a cavity behind. The spinal cord is somewhat swollen throughout its entire extent, and on section the transverse markings are seen to be greatly blurred. Hayem describes this form in the following words: diffuse inflammation of the gray substance, more or less diffuse hyperæmia of the white substance; small, band-shaped foci of inflammation in the latter; more or less intense, meningitic changes.

When this form possesses a hemorrhagic character, we have the *myelitis centralis hæmorrhagica*, which Hayem designates by the term *hæmatomyelitis*. It is characterized by a more or less abundant extravasation of blood, which appears either in the form of a marked hemorrhagic softening, affecting chiefly or exclusively the gray substance, or in the form of hemorrhagic foci of varying sizes, which may extend into the white substance. The tissue in the neighborhood of these foci, both above and below, is in a state of myelitic softening, that may extend throughout almost the entire length of the medulla.

The *acute myelitis without softening*, which Dujardin-Beaumetz terms *myelitis hyperplastica*, also deserves special mention. Its essential characteristic is an increase of the interstitial tissue, which becomes unusually dense and very rich in nuclei. The septa are wider than usual, and the vessels are thickened; there are a few granular cells. The consistence of the cord is normal or somewhat increased. This form seems to occur principally in small spots (Westphal's myelitis disseminata), and to indicate in general a slighter degree of intensity in the inflammatory process. It is sometimes observed also in the neighborhood of spots of acute softening. It should probably be classed rather with the subacute forms, which constitute an intermediate step between acute and chronic myelitis.

The changes in the other organs of the body, in acute myelitis, require only a brief enumeration. In the peripheral nerves we find in many cases—especially in central myelitis—degenerative atrophy in its different stages of development. In the muscles supplied by these nerves, we find also the characteristic signs of the first stages of the same process (proliferation of the nuclei, slight atrophy of the fibres, etc.).

The mucous membrane of the urinary bladder is in many cases swollen, flecked with hemorrhages and in a state of catarrh; in the severest cases it is infiltrated with diphtheritic exudation and presents foul, sloughy ulcers. Sometimes similar changes are observed in the ureter extending into the pelvis of the kidney, and in not a few cases the kidneys have been found filled with metastatic abscesses. According to Hayem, a part of these originate from collections of vibriones.

The changes characteristic of decubitus acutus in its different stages of development, are usually observed on the skin covering the sacrum and the nates, the trochanters, and other spots exposed to mechanical pressure. (Compare Part I., p. 121.)

Other pathologico-anatomical changes, which are more or less accidental, do not require special consideration here.

Pathology of Acute Myelitis.

Symptoms.

The *general clinical history* of acute myelitis is so manifold and changeable, and so different perhaps in each individual case, that we can only attempt to give here its most common features. Later on these will be filled out in many particulars while considering the symptoms in detail.

Even in its *mode of commencement* the disease is very variable. Sometimes it is preceded by general malaise and slight febrile movement, while in a few cases it commences with a chill, which is followed by the usual febrile symptoms: headache, general depression, dragging pains in the limbs, loss of appetite, etc. Frequently the scene is opened at once by *spinal symptoms*. In these cases the symptoms which first attract the attention of the patient are usually *disturbances of sensation:* paræsthesiæ, formication, etc., in the extremities, a girdle-sensation or perhaps a girdle-pain at the level of the seat of disease, pain in the back and tenderness over certain spinous processes, sometimes painful, dragging sensations in the bladder and rectum, gastralgic pains and the like. These are sometimes, but not always, accompanied by *motor symptoms of irritation:* twitchings of certain muscles or groups of muscles, violent tremor, partial clonic spasms and in some special cases even general convulsions.

Sometimes an isolated paralysis of the bladder is the first symptom.

Very soon, however—sometimes after a few hours, or on the next day—the *characteristic paralytic symptoms* make their appearance. Of these, the *motor paralysis* is the first to appear; it is more or less complete and involves a variable number of muscles, usually taking the form of paraplegia, but not unfrequently assuming other forms.

Paralysis of the sphincters usually sets in soon after this. It often appears very early, simultaneously with the motor paralysis, but in some cases it does not occur until long afterwards. It may, however, be entirely wanting.

The same may be said of the *paralysis of sensation.* In all severe cases it sets in very early and is complete; there is then usually complete anæsthesia of the lower half of the body up to a certain height, which is sometimes marked by a pretty sharply defined line. In milder cases the anæsthesia may be less complete and less extensive, and finally in certain forms of the disease (poliomyelitis anterior), it is entirely wanting.

The *reflex excitability* varies according to the seat of the disease. It may be rapidly and completely destroyed in the paralyzed part, or it may gradually diminish as the disease progresses, or it may remain normal, or finally it may be very considerably increased, occasionally attaining an astonishing degree of intensity.

For most cases of acute myelitis the *rapid extension of the paralysis* through the cord in a horizontal direction, the complete paraplegia, is to some extent characteristic; not less so in many cases, is its rapid extension in a vertical direction, the rapid ascent of the paralysis towards the head, the acute ascending paralysis. These peculiarities depend on the variety and localization of the disease in the individual cases. In sharp contrast to them, there are others in which the paralysis attains its acme in the very commencement, never extending subsequently.

In almost all the severe cases, vaso-motor paralyses, cystitis and pyelonephritis, and extensive bed-sores which are often acute, set in early. All these complications may, however, be absent.

The same is true of the atrophy of the paralyzed muscles, and of the loss or modification of their electrical excitability.

The subsequent *course* of the disease varies greatly in the different cases and forms:

1. It may be *rapidly progressive and fatal.* In this case, after a few days of continuous fever with the symptoms of an ascending paralysis, death takes place from a rapidly developing asphyxia.

2. Or the disease may run a *slower and less violent course;* the fever remains moderate or disappears entirely, but cystitis and bed-sores are developed, bringing in their train fever, emaciation and exhaustion, and the patients succumb after weeks or months of suffering.

3. Or the acute affection may be *transformed into chronic myelitis;* the paralytic symptoms persist with slight variations in intensity, until at some later period a slow exacerbation sets in, and the affection then runs the usual course of a chronic myelitis.

4. Or an *improvement sets in,* which, however, *always remains incomplete.* The myelitic process ceases, but not until it has irreparably destroyed a certain number of the nervous elements of the cord, as a result of which a more or less extensive defect, such as paralysis, atrophy, contracture, anchylosis, or the like, is left behind. The general health, however, becomes and remains good, and the disease which he has passed through exerts no further influence on the length of the patient's life.

5. Or finally—and this is perhaps the rarest termination of all —*complete recovery* takes place. Several reported cases demonstrate that this favorable termination is at all events possible. A rapid improvement sets in, and, after the expiration of a longer or shorter period, all the bodily functions are completely restored. Convalescence is usually somewhat protracted.

As has been stated, this clinical picture, which contains only the most general features of the disease, can be modified in many ways, the necessary result of which has been the differentiation of various forms of acute myelitis, to which we will return later. Before doing so, however, we must describe somewhat more closely the *individual symptoms.*

Among the *disturbances of sensation* the *symptoms of irritation*, in consequence of the complaints of the patients, usually assume the greatest prominence. In a few rare cases they are entirely wanting, or are so slight that the patients do not speak of them, unless closely questioned about them. Violent shooting pains are not commonly present in acute myelitis, a point of importance in the differential diagnosis from meningitis. They do occur, it is true, especially in the initial stage, but usually disappear soon, seldom persisting for any length of time. They appear sometimes in the form of neuralgic pains surrounding the trunk like a girdle at a variable height, sometimes as a circular, hoop-like, burning sensation in the skin, or they may consist of dragging, tearing, boring or burning sensations in the limbs. Pressure and movement do not usually aggravate these pains in the extremities. They are very commonly accompanied by pain in the back, extending over a more or less extensive area, and also by tenderness over several of the spinous processes. This can sometimes be recognized by passing hot and cold sponges over the spine of the back. On the other hand, various paræsthesiæ belong to the more constant symptoms of the disease. One of them is the well-known unpleasant sensation of constriction as by a girdle, which is experienced not only on the trunk, but also in the extremities and joints. Feelings of tension or swelling, and of cold or heat, pricking and sticking sensations, formication, etc., are experienced over more or less extensive cutaneous areas, particularly in the lower extremities. Gradually the feeling of numbness and furriness and the loss of sensation become more and more prominent as the anæsthesia becomes more distinct. True hyperæsthesia is rarely present in acute myelitis, and when it is, is probably due usually to a complication with meningitis. It does, however, occur also in unilateral circumscribed myelitis, on the same side as the motor paralysis. Charcot describes, under the name of dysæsthesia, a peculiar, diffuse, painful, vibrating sensation, which is produced by touching any circumscribed spot on the skin, and may extend to the entire extremity and even to both lower extremities.

The *symptoms of sensory paralysis* are not usually slow in making their appearance, though they too may be permanently absent. The anæsthesia may be more or less extensive and complete; the paralysis of sensation is sometimes only partial, or there may be merely a slowness in the conduction of sensations. Severe pains are not unfrequently complained of in parts that are entirely deprived of sensation—anæsthesia dolorosa. Isolated, jerking, shooting pains, accompanied by spasmodic twitchings of the muscles, are very common in the paralyzed parts.

The explanation of all these symptoms is undoubtedly to be sought in the development and progress of the inflammatory changes in the spinal cord. The initial symptoms of irritation are to be ascribed to the inflammatory and hyperæmic irritation of the nerve-elements in the gray and white substances, the later symptoms of paralysis to the destruction of these elements and their compression by the inflammatory exudation. The girdle-sensations depend probably on the involvement of the posterior nerve-roots in the inflamed spot; the paræsthesiæ and anæsthesiæ of the posterior half of the body, on the involvement of the sensitive tracks situated in the gray substance and in the posterior white columns. Since these sensitive tracks lie for the most part within the gray substance, or at all events must pass through it for a certain distance, it is easy to account for the usually intense and complete anæsthesia in acute central myelitis. Further, the well-grounded assumption, that the sensitive tracks run principally or exclusively in the posterior half of the gray substance and the posterior and lateral white columns, explains sufficiently the fact, that in myelitis involving only the anterior portion of the gray substance (*e. g.*, in the spinal paralysis of children), disturbances of sensation are entirely wanting. In the circumscribed, disseminated, and peripheral forms of myelitis, the degree of the disturbance of sensation will depend solely on the extent to which the sensitive tracks are involved in the pathological changes in the cord.

The *motor disturbances* also present irritative and paralytic stages. To the former belong, in the first place, the twitchings of individual muscles or of entire extremities, which often occur in the initial period, and the spasmodic contractions of the muscles, which sometimes increase to a condition of tetanic rigidity;

also the convulsive movements of the extremities, and finally the initial general convulsions, which occur almost solely in children. Much more constant and more important, however, are the *symptoms of motor paralysis*, the development of which stamps the clinical picture of acute myelitis with such a characteristic impress. The paralysis may be developed with such enormous rapidity, that we speak of an apoplectiform myelitis. Cases have been observed, in which complete paraplegia was developed during one night, within a few hours, and even in less than an hour. It is, however, only in the hemorrhagic form of myelitis that the paralysis can develop fully within the space of a few minutes, just as it does in apoplexy of the cord. This rapid development is the rule particularly in central myelitis; in the other forms of the disease, a longer period may be necessary for the production of the paralysis. In such cases the muscles are always perfectly flaccid; the limbs hang or lie as if they were dead, and when raised fall like the limbs of a corpse.

Later in the disease, if the patient survive, the *symptoms of irritation* may again make their appearance in the affected limbs. Isolated spontaneous twitchings of the muscles are observed, which are often accompanied or excited by severe shooting pains. A state of muscular tension, or spasmodic tonic contractions of the muscles set in, which are excited particularly by attempts at voluntary movement or by irritation of the sensitive nerves. Finally, severe contractures are produced, which fix the legs in an extended or flexed position, and are frequently rendered more intense by attempts at active or passive movements. These symptoms, however, belong more to the later periods of the disease, and hence are more constant in the subacute and chronic forms.

The most common form of the paralysis is paraplegia; still hemiparaplegia, paraplegia cervicalis and isolated paralyses of particular limbs, as well as complete paralysis of all four extremities along with the trunk, also occur. This depends, of course, entirely upon the seat and extent of the disease.

The pathogenesis of all these motor disturbances is not by any means perfectly clear as yet. There can be no doubt, it is true, that they must be due to changes in the motor nerve-tracks in the spinal cord, but we are unable to decide positively in every case, whether they are due to lesions of the anterior roots, or of the nerve-tracks in the gray substance or in the lateral columns, or to simple reflex processes. The arguments that are advanced in support of each of these suppositions have already been detailed while speaking of the general disturbances of motility (see Part I., pp. 79 and 97), and to avoid repetition we may refer the reader to that chapter. It is consequently in regard to the localization of the morbid process, that the explanation meets with the greatest difficulties, for it is a self-evident fact that the inflammatory changes in the cord must be capable of producing irritative as well as paralytic phenomena in the motor paths.

The *disturbances of reflex action* are especially valuable for the interpretation and localization of the lesion in many cases of acute myelitis. The state of the reflex activity, both of the skin and the muscles, may vary greatly; it may be diminished and even destroyed, or it may be considerably increased. In a portion of the cases it is abolished very soon, immediately after the development of the paralysis, and reflex actions can no longer be excited even by severe irritants; sometimes it is not entirely abolished, but a longer time is required for the production of the movements; in other cases it undergoes a very considerable increase, so that even very slight irritations call forth the most active, reflex muscular twitchings, which may increase to a persistent, convulsive jerking of the paralyzed part; or, in still other cases, it remains unaltered or is slightly increased for a time, and then begins to diminish and gradually becomes weaker, finally disappearing entirely. Whatever the condition may be, it furnishes a valuable indication of the state of the gray substance, for it is this portion of the cord which determines principally the state of the reflex activity. The more intact the gray substance remains, the more confidently can we look for a continuance of the reflex actions. When a portion of the gray substance, itself intact, is separated from its connection with the brain by a more centrally situated myelitis (*e. g.*, the gray substance in the lumbar portion of the cord by a myelitis located in the dorsal region), the reflexes are increased; hence the exaltation of the reflex excitability so commonly observed in myelitis transversa dorsalis. When the gray substance is destroyed by the disease the reflexes are abolished; hence their disappearance in myelitis of the lumbar enlargement, and more especially in diffuse central myelitis, in which all reflex action is usually abolished from the very commencement. When this destruction of the gray substance is secondary and effected at a later period of the disease by the extension of the inflammation downwards, the reflex activity may be at first normal or even increased, but will subsequently become weaker and gradually disappear.

Hence, we shall be able in many cases to draw from the condition of the reflex excitability important and relatively sure conclusions, with regard to the localization of the myelitis in the gray substance. We must bear in mind, however, that the reflex actions can also be modified by changes in the anterior roots, and that, moreover, the lateral columns probably exert an important influence on them—facts which warn us not to make these conclusions too positive.

The *sphincters* are very frequently involved. Vesical paralysis is not unfrequently one of the earliest, and sometimes even one of the prodromal symptoms of acute myelitis. In severe cases there is usually complete retention of the urine, which must be evacuated by the catheter during the entire course of the disease. In other cases there is merely incontinence of urine, or this is the most constant symptom on the part of the bladder. In a word, any of those disturbances which we have described and attempted to explain in Part I., p. 132, may be observed. Sometimes the symptoms in the beginning of the disease are more those of irritation, spasmodic closure of the sphincter with increased desire to make water.

The sphincter ani behaves in a similar manner; here, too, the most constant symptoms are those of paralysis. The explanation of all these symptoms on the part of the sphincters is to be found in the localization of the myelitis; for further particulars we may refer to what has been said on this subject in the general part.

Priapism is not an uncommon symptom in acute myelitis.

Generally the erection of the penis is incomplete, but it often persists for days, with slight variations in degree. It must be regarded as essentially a sign of irritation, produced either by irritation of the nerve-tracks passing from the brain to the lumbar portion of the cord (Comp. p. 313), or by reflex excitations proceeding from the bladder, skin, etc.

The *vaso-motor disturbances* in acute myelitis have not yet been sufficiently studied. Engelken found in one case a rise in the temperature of the paralyzed part, but most authors speak of the extremities as being cold. Diffuse œdema of the paraplegic lower limbs has sometimes been observed. The perspiration is sometimes increased, sometimes diminished in quantity. As recent physiological investigations have demonstrated that the vaso-motor innervation is of a complicated nature, we must wait for more exact observations before an explanation of these disturbances can be attempted.

This applies with even more force to the *trophic disturbances,* which follow in the train of acute myelitis. They are most distinct in the skin, the most striking of them being the decubitus acutus,[1] which is developed regularly and early in the severer forms of myelitis, and occasions great danger to life. It appears in the well-known form of gangrenous inflammation of the skin over the sacrum, trochanters, etc., usually progresses rapidly, and proves fatal by septic fever. When the lesion in the cord is unilateral, the decubitus is situated on the opposite side of the body. It may make its appearance as early as from the second to the fifth day of the disease. For its pathogenesis we may refer to the remarks made in Part I., p. 123.

There is not always time for the development of trophic disturbances in the *nerves* and *muscles;* still, even in rapidly fatal cases of central myelitis, the first traces of the degenerative atrophy have been discovered in the muscles and nerves. When the disease has been of somewhat longer duration, the atrophy of the muscles is not unfrequently very marked. There seems to be no question that these disturbances in the nutrition of the muscles are directly dependent on the condition of the gray substance. Every marked disturbance of nutrition in the gray substance leads to rapid atrophy of the muscles, and the study of particular forms of acute myelitis (spinal paralysis of children) has demonstrated very positively that this influence on the nutrition of the muscles is exerted especially by disease of the anterior horns. Hence, wherever a considerable and rapidly developed atrophy of the muscles exists, we must assume an inflammation of the gray matter.

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[1] TRANSLATOR'S NOTE.—The Germans use the term decubitus to designate bed-sores, and for the sake of convenience it has been deemed advisable to retain it in the translation.

The state of the *electric excitability* is of great value for the recognition of this degenerative atrophy. Whenever rapid muscular atrophy sets in, or in other words in severe affections of the gray substance, a loss of the faradic irritability of the nerves and muscles is also observed; and, unless greatly deceived, we may look for the development of the reaction of degeneration (Entartungsreaction) in all such cases. Death, it is true, often occurs so rapidly, as to leave no time for its complete development. In the more subacute cases, however, we shall frequently be able to demonstrate this reaction of degeneration, and in the forms of acute myelitis in which the changes are confined to the anterior horns of the gray matter, it is one of the most constant phenomena. On the other hand, there are cases of acute myelitis in which there is absolutely no change in the electric irritability, and others in which only slight quantitative changes in the same, slight increase or diminution, can be demonstrated. The conclusion, that in such cases the corresponding portions of the gray substance have remained intact, cannot be questioned.

Marked *alterations in the urinary excretion* occur with striking rapidity in severe cases of acute myelitis. After a very few days (two to eight), the urine is alkaline and not unfrequently bloody; a muco-purulent deposit, numerous triple-phosphate crystals, etc., form very rapidly. It is not improbable, though by no means proven, that these alterations are directly dependent on the acute spinal affection, and not merely the result of the retention of urine caused by the paralysis of the bladder.

Cerebral phenomena are not regularly or even frequently present in acute myelitis. Still they are not exactly rare in the commencement of some particular forms (poliomyelitis anterior), though they usually last only a short time. In children especially, we observe in such cases headache, delirium, and general convulsions. It is scarcely possible to determine, however, what part in the production of the last-named symptom is played by the great irritability of the central nervous system of the child, and what part by the disease of the spinal cord.

The headache, delirium, etc., in acute myelitis may also occasionally be due to the fever, or to a complicating septicæmia or uræmia.

Graver cerebral symptoms make their appearance, when a process analogous to that in the cord is established in the brain, where it produces its characteristic local manifestations.

Oculo-pupillar symptoms are sometimes developed in cervical myelitis. Speaking and swallowing may also be interfered with, when the disease involves the medulla oblongata.

The disorders of the optic nerve, of the nerves supplying the muscles of the eye and of other cerebral nerves, which play a very prominent rôle in the clinical histories of many cases of chronic myelitis, are unknown in acute myelitis.

On the part of the *digestive organs*, the most constant symptom is great obstipation, for the relief of which the most energetic remedies are often required. It is probably due to paralysis of the muscular coat of the intestines. Later on this may lead to great meteorismus, which may become so excessive as to be dangerous to life. In the commencement of the disease violent paroxysms of cardialgic pain are sometimes observed; their pathogenesis is undoubtedly the same as that of the girdle-pains.

Little is known concerning the behavior of the *circulatory organs*. The pulse is usually increased in frequency. When the myelitis extends to the cervical portion of the cord, the increase in the frequency of the pulse may be very great, and finally a state of pronounced cardiac weakness is produced. Many patients suffer much from palpitation or irregularity in the action of the heart, which is accompanied by unpleasant, twitching sensations in the cardiac region.

The *disturbances of the respiration* are better understood and more important. When the myelitis is primarily located in, or has extended to, the cervical region of the cord, they occupy the foreground in the clinical picture, and occasion immediate danger to life. In cases of ascending central myelitis, the gradual encroachment on the respiratory movements, terminating finally in complete paralysis, can be followed with great facility. First of all, the abdominal muscles are paralyzed, and expiration and expectoration are impeded. Next in order, the intercostales and the muscles of the trunk are affected; as a result inspiration is interfered with, and the patients breathe only with the diaphragm. When this last-named muscle is also involved in the paralysis, the dyspnœa becomes very intense, inspiration being carried on only by means of increased activity of the cervical muscles, and death by asphyxia very soon occurs in consequence of paralysis of the respiratory centres. In this way the fatal termination can be brought about with very great rapidity in severe cases, death often occurring in an acute attack of dyspnœa. Other cases, however, are more protracted, and the paralysis is then usually complicated by pulmonary hypostasis. The pathogenesis of the respiratory disturbances requires no special elucidation.

The *general health* is greatly impaired in almost all cases. In exceptional instances the appetite is not affected and the patients sleep well, and the nutrition of the body consequently remains tolerably good. Usually, however, the fever, the psychical depression, the commencing cystitis and the decubitus occasion sleeplessness and loss of appetite which are soon followed by marked disturbance of nutrition, great emaciation and finally exhaustion.

In the commencement of acute myelitis, *fever* is present only in a small number of cases. In these cases, however, it may at once become very high, and it may remain persistently high throughout the entire course of the disease. Sometimes it occurs in isolated, sharp attacks, and an excessive rise of temperature is not infrequently observed immediately before death. In other cases the fever is slight, never attains a high grade, and may even disappear entirely during the subsequent course of the disease. To sum up, the fever in acute myelitis is in no respect characteristic. As might be expected, the cystitis, pyelonephritis, decubitus and septic infection very frequently give rise in the later stages of the disease to a symptomatic fever, which exhausts the strength of the patients.

Course, Duration, Terminations.

With regard to the *course* of the disease but little can be said that will be generally applicable, on account of the great divergences presented by the individual cases. This fact is sufficiently evident from what has already been said, and it will be rendered still more apparent, when we come to speak of the different forms of the disease.

The abrupt commencement of the disease in almost all of the cases, and the speedy development of the symptoms up to a certain point, are to some extent characteristic of acute myelitis. It is the rapid development of the paralytic phenomena, rather than the presence and the degree of the fever, which usually leads us to characterize a myelitis as "acute." It is not exactly necessary for the paralysis to be developed within a few hours, in order to justify the employment of the term "acute," but it must at all events make its appearance within a few days (about one to ten). Cases which run a slower course may be classed as subacute, but no sharp line of demarcation can be drawn between the two forms.

In not a few cases the commencement of the disease is exceedingly abrupt. The paralysis may develop in an apoplectiform manner, almost without premonitory symptoms, and it may attain considerable intensity in an hour or even less. Often, patients awake in the morning with complete paraplegia, who had retired to bed on the preceding evening feeling scarcely at all unwell (myelitis apoplectica). Usually, however, there is a prodromal stage of variable length, and after the paralysis has commenced, hours or days elapse before it develops into pronounced paraplegia. In some cases, which must also be classed as acute myelitis, a number of days elapse before this point is reached. Sometimes the development of the disease, instead of being continuous, is interrupted by successive pauses.

Once begun, the subsequent course of the disease is also variable. In the most violent and rapid cases (myelitis centralis, hæmatomyelitis, etc.) the paralysis rapidly ascends, symptoms of asphyxia appear, and death takes place in *a few days;* or the fatal termination is brought about by the violent fever and septicæmia caused by the acute cystitis and decubitus, and occurs after a somewhat longer period, at most *after a few weeks.*

In the less severe cases, particularly when the entire lumbar enlargement with or without the dorsal portion of the cord, is affected, the course is somewhat slower. There is complete paraplegia with paralysis of the bladder; cystitis is gradually developed, followed by decubitus, fever, cachexia and exhaustion, and the patient succumbs *after several weeks or months.*

In still other cases *the transition to chronic myelitis* is effectuated. There is then usually complete paralysis of motion, with incomplete paralysis of sensation and paralysis of the bladder. The symptoms remain stationary for months and years, or present slight oscillations, being at one time better and at another worse. Cystitis and decubitus may develop after the disease has persisted for a variable time, but they never become very severe, and are susceptible of being at least partly cured. Death finally results from exhaustion or from some intercurrent disease.

In other cases, again, the disease ceases, leaving only a few unimportant and harmless traces of its passage. The disorders of sensation and of the bladder, and the trophic disorders of the skin are usually entirely wanting in these cases. The general health is soon completely restored. The only traces left by the disease are paralysis and atrophy of one or more muscular groups with their consequences, the patients feeling in all other respects perfectly well. The duration of life is not affected in any way by the myelitis. This is the *termination in imperfect recovery.*

Finally, *complete recovery* takes place in some very rare cases. In consequence of the lack of autopsies, however, it will always be possible for sceptics to question the correctness of the diagnosis in these cases. This complete recovery probably takes place only in the milder forms of myelitis, though these are not unfrequently quite diffuse. This termination usually sets in early.

After the symptoms of paralysis, the fever, etc., have persisted for one or two weeks, they undergo a slow and gradual retrogression, and after the expiration of a few weeks all the functions of the body are completely restored. Convalescence is, however, almost always very protracted. Medical literature contains a number of reported cases which demonstrate pretty positively the possibility of this termination.

It still remains for us to point out briefly the characteristics of the *different forms of acute myelitis*, as they most frequently come under observation.

The traumatic myelitis, which develops after severe injuries of the cord, can be regarded as the type of acute *myelitis transversa.* It is most frequently located in the dorsal region of the cord. Its principal symptoms are: girdle-sensation and girdle-pain, complete paraplegia, anæsthesia, paralysis of the bladder, preservation and increase of the reflex excitability, absence of muscular atrophy, retention of electric irritability; in the later stages, symptoms of motor irritation, contractures, etc.; also cystitis and decubitus. The persistence of the reflex excitability is particularly important.

Acute *myelitis centralis*, as described especially by Dujardin-Beaumetz and Hayem, includes the cases which run the most rapid course. The commencement is usually abrupt and attended by disturbances of sensation; complete anæsthesia and paralysis of the lower half of the body with entire relaxation of the joints, are developed very rapidly, often in the course of a few hours or during a night; paralysis of the bladder and rectum. The extinction of all reflex excitability and the early commencement of progressive muscular atrophy, with loss of faradic irritability, are specially important. Later on, decubitus acutus, alterations in the urine, often œdema of the paraplegic limbs and neuropathic articular affections, more or less intense fever, progressive advance of the paralysis upwards, early death by asphyxia.

The hemorrhagic form of myelitis centralis, or *hæmatomyelitis* (Hayem), does not differ essentially from the simple form in the commencement. It runs the same rapid course, but the very abrupt appearance of the paralysis, which becomes complete in the course of a few minutes, or at most of an hour or a little more, marks the occurrence of hemorrhages. The more rapid the development of the paralysis, the more predominant is the hemorrhagic element in the process. In other respects the course is the same as in the simple form.

Hemorrhagic myelitis can only be distinguished from simple, idiopathic hemorrhage into the substance of the cord, in those cases in which the hemorrhage is preceded by pronounced symptoms of an acute myelitis; pains, paræsthesiæ, girdle-sensation, vesical weakness, muscular twitchings, commencing paresis, fever, etc. (Compare p. 291.)

The so-called *poliomyelitis anterior acuta* (Kussmaul),. the spinal paralysis of children, which occurs in isolated, circumscribed spots in the anterior horns of the gray matter, is clinically a sharply characterized form of acute myelitis. Acute febrile commencement, often with headache, delirium and convulsions; rapid development—in a few hours or days—of motor paralysis, which varies in area in different cases, but attains its maximum of extension in the very beginning; no disturbances of sensation, no paralysis of the sphincters, no decubitus; on the other hand, rapidly progressive muscular atrophy, with the reaction of degeneration; eventually, atrophy and impairment of the growth of the bones, deformities of the joints, etc. Complete restoration of the general health, early improvement of the paralysis up to a certain point, a number of the muscles, however, almost always remaining atrophied and paralyzed, causing permanent deformities. (Compare Section No. 15.)

In *myelitis disseminata*, as described by Westphal, the clinical picture is naturally not so characteristic. It ordinarily causes paraplegia, sometimes with spastic symptoms, usually with paralysis of the bladder. The state of the sensibility is variable; it is sometimes more, sometimes less impaired. The same statement holds true of the reflex and also of the electric excitability. Sometimes the existence of several centres of disease can be recognized from the grouping of the symptoms, the exacerbations, etc. The development of the symptoms after acute diseases, especially variola, or in phthisical patients, etc., might direct suspicion to this form of the affection.

Acute myelitis is very frequently complicated with meningitis, *myelomeningitis acuta.* The addition of meningitic changes to the myelitic affection, although unquestionably very common, is not specially important, since the severity and dangerousness of the disease is not essentially modified thereby. The development of myelitis as a complication of acute meningitis is a more serious affair. We have already, in another place (p. 226), spoken of this condition of affairs, and endeavored to form an estimate of its influence on the symptomatology of acute spinal meningitis; this influence is, at all events, greater than has been hitherto supposed. Finally, the two inflammatory processes can set in simultaneously, so that neither one of them is dependent on or secondary to the other. Under all these conditions, the meningitic symptoms usually assume the most prominent rôle in the subjective complaints of the patients, while the myelitic symptoms are the most striking in the objective examination. In such cases the phenomena which speak for meningitis are chiefly the pain and stiffness in the back, the cervical rigidity, the pronounced hyperæsthesia, the diffused pains, etc., while the contractures, the severe paralytic symptoms, the paralysis of the sphincters, the augmented reflex activity, etc., must be placed more to the account of the myelitis. More exact observations and investigations are, however, necessary for the clearer differentiation of the two processes.

Diagnosis.

In the matter of diagnosis, acute myelitis resembles many other affections of the spinal cord; the typical cases are easily recognized, while on the other hand, the less pronounced, the complicated and the uncommon cases are difficult to unravel.

It is easy to gather from the preceding description, what the characteristic symptoms are, from which an acute myelitis can be easily diagnosticated; acute commencement with more or less marked signs of sensitive and motor irritation, very rapid development of complete paralysis, vesical paralysis, bed-sores, etc. When fever is also present, and the etiological conditions are known, the diagnosis is easy.

There are several other diseases with which it may possibly be confounded. First of all we must mention *paralysis ascendens acuta* (see farther on, No. 17), which resembles diffuse central myelitis in particular so closely, that it is often scarcely possible to determine to which of the two diseases the case in question belongs. The results of recent investigations (Westphal) have gradually forced us to the conclusion, that paralysis ascendens acuta is a special form of disease, entirely distinct from myelitis. The diagnostic points, which speak rather for acute ascending paralysis, are the following: absence of convulsive movements at the outset of the affection, absence of trophic disturbances, a slight degree of encroachment on sensation and preservation of electric irritability. In many cases, however, the positive diagnosis can only be made with the help of the autopsy.

The difficulty of distinguishing acute myelitis from *meningitis acuta* is often very great, in consequence of the frequent combination of the two diseases. The following symptoms speak for meningitis: high fever, severe pain, dorsal and cervical rigidity, contractures, slight symptoms of paralysis, especially on the part of the sphincters, absence of severe trophic disturbances, pronounced hyperæsthesia, etc. (Compare also p. 245.)

The differentiation of *hæmatomyelia*, or simple hemorrhage into the spinal cord, from central myelitis, and particularly from the hemorrhagic form of that affection, is often uncommonly difficult. The diagnostic points are: the *very abrupt* development of the paralysis without fever or prodroma, the stationary character of the paralysis, etc. This question has already been fully discussed in another place (p. 303).

The differentiation of myelitis from *hæmatorrhachis*, or hemorrhage into the meninges of the cord, is, as a rule, easier. The very abrupt development of the affection without premonitory symptoms or fever, the symptoms of severe meningeal irritation, the violent pains, the dorsal rigidity, the relatively moderate grade of the paralytic phenomena, and particularly the slight intensity of the anæsthesia, usually characterize hæmatorrhachis sufficiently. (See p. 214.)

It is scarcely possible to confound acute myelitis with *hyper-*

æmia of the cord. The absence of fever, the slight intensity of the sensory and motor disturbances, the frequent and rapid variations in the symptoms, and the absence of vesical weakness and of bed-sores, are so characteristic of the latter affection as to almost prevent the possibility of a mistake.

The diagnosis from *hysteria* can scarcely present any difficulties; at all events any doubt which may arise, will be cleared up by a few days' delay. It is unnecessary to recapitulate here the points of the differential diagnosis. On the other hand, many poisons produce symptoms, which may resemble the clinical picture of ascending central myelitis so closely, that the differentiation is very difficult.

The *seat* of the disease in the cord and its *extension* in the transverse and vertical directions, can readily be determined from the area of the paralytic phenomena, the state of the reflex irritability, the trophic disorders, etc. As these points have already been repeatedly discussed, we may dispense with further consideration of them here.

Prognosis.

The *prognosis* varies vary greatly. In general it is unfavorable, because the disease is almost always severe. There are, however, numerous exceptions to this rule.

A perfect recovery is rare. In many cases a chronic state of disease persists, while in others the morbid process ceases, but leaves a legacy of incurable, although relatively harmless defects. The prognosis, at least as far as life is concerned, is often absolutely favorable (poliomyelitis anterior acuta).

This much being premised, we may state that the prognosis depends chiefly on the *location and extent of the inflammatory process.* It is not correct to say, that it always becomes worse the higher the location of the disease in the cord. Strictly speaking, that assertion holds true only when the disease is located in the dorsal and cervical regions; in such cases the higher up it is located, the greater is the liability of the respiratory tracts becoming affected, hence the increasing danger to life. On the contrary, a myelitis in the dorsal region is, *ceteris* *paribus*, more favorable than one in the lumbar region, on account of the important centres located in the latter.

It is much more correct to say, that the prognosis depends on the *extent of the cross section of the cord involved in the process;* the more considerable this is, the more unfavorable is the prognosis. It becomes worse in proportion especially to the extent of the gray substance involved. The central and posterior portions of the gray substance seem to be particularly dangerous in this connection, for experience teaches that acute myelitis of the anterior horns alone is not attended by any danger to life, although it certainly annihilates the function and the nutrition of the muscles. In myelitis of the central gray substance, on the other hand, the prognosis is much more unfavorable, on account of the usually unavoidable development of cystitis, decubitus, etc. We are as yet unable to say how much the more or less extensive implication of the white columns influences the prognosis.

The *extent of the longitudinal section of the cord involved in the processes* influences the prognosis in a similar manner; it becomes worse in a direct ratio to the longitudinal extent, or, in other words, to the length of the diseased spot. Hence a very circumscribed, transverse myelitis is not so dangerous as the same affection, when it extends over a greater length of the cord. Hence, also, the unfavorable prognosis in the progressive, ascending forms of the disease, and particularly in central ascending myelitis. A longitudinal extension of the disease in the white columns is, however, not by any means so portentous.

There are various other facts which can also be made use of for the prognosis. A very rapid development and great intensity of the paralysis, complete paralysis of the sphincters, early and especially acute decubitus, progressive advance of the disease upwards, high fever, great impairment of the general health, disorders of respiration, dyspnœa, cyanosis, etc., influence the prognosis *unfavorably*. On the other hand, a moderate degree of paralysis, absence of trophic and sensory disturbances, implication of the bladder to only a slight extent, absence of fever and of marked impairment of the general health, commencing improvement of some of the symptoms, etc., are signs of *favorable portent.*

The nature of the etiological influences and the possibility of removing them, the possible occurrence of exacerbations and relapses, the general condition of the patients, the effects produced by certain therapeutic measures, etc., must also be taken into account in determining the prognosis.

By close attention to all of these points, we shall in many of the cases succeed in forming a tolerably correct judgment of the course and termination of the disease.

Treatment.

Of a *prophylactic treatment* of acute myelitis, there can hardly be any question. If there be any it consists simply in the observation of those general rules, which are in any case necessary for the preservation of health.

On the other hand, a *causal treatment* is possible in many cases. Injuries of the spinal column must be treated surgically, and simple concussions must also receive proper attention. Tumors of the spinal column should be removed if possible; in these cases the acute meningitis also requires treatment. When the premonitory signs of myelitis make their appearance after exposure to cold, etc., the morbid process can be perhaps arrested or mitigated by an energetic diaphoretic treatment (diaphoresis, hot drinks, the warm bath, the pack, derivation to the back, etc.). Where there has been excessive bodily exertion, the same end may perhaps be attained by absolute rest in the proper position, a warm bath, etc. Suppressed secretions, profluvia and hemorrhages should be excited afresh, if there be any reason to suspect that the suppression has had any influence in causing myelitis. The treatment of syphilis is of special importance, when an acute myelitis is developed in its course; in such cases an energetic mercurial treatment must be immediately commenced and be supplemented by large doses of iodide of potassium, "Roob Laffecteur," etc., when the stage of the disease indicates them.

As a rule, however, the patients do not come under observation until the disease is developed. The measures to be adopted then will of course vary both in nature and in activity, according to the form and the severity of the disease. The general features of these measures of treatment are all that we can attempt to describe here.

In all cases that are at all severe and threatening, an *energetic antiphlogistic treatment* is indicated. This is not the place to discuss the justifiableness of the usual antiphlogistic measures. We are, moreover, still very much in want of actual observations on which to base a reliable opinion as to their efficiency in acute myelitis. Nothwithstanding this, however, in the face of so dangerous a disease, the energetic employment of all antiphlogistic measures is decidedly indicated. Active *local bloodletting* from the spine may be practised by means of leeches or wet cups, applied several times in succession; Chapman's *ice-bag*, from which Tibbits saw very good results, may be employed; the trunk may be enveloped in, or *the back covered with, cloths wrung out in water*, which should in turn be covered with caoutchouc-paper and flannel, and should be renewed every few hours; to these may be added the *inunction of mercurial ointment* into the back and other parts of the body—a measure of doubtful utility, but still one which is permissible in view of its efficiency in inflammatory affections of the eye, even when not of a specific nature. For the same reason, small doses of *calomel*, frequently repeated, may be tried, and eventually also *iodide of potassium*, in sufficient doses. Brown-Séquard recommends, on the ground of physiological experiment, the employment of *ergot* and *belladonna* (to combat the concomitant hyperæmia); these remedies deserve a trial.

In the majority of the cases, however, a moderate *derivation to the intestines* (castor oil, "aqua laxativa," mineral waters containing sulphate of magnesia, senna, rhubarb, colocynth, etc.), and *also to the kidneys* (by the ingestion of large quantities of water, the use of Ems or Vichy water, of acetate of potash, etc.) is more important and more useful. Attention must be paid to this.

One difficult question which presents itself in the treatment of acute myelitis is, whether or not the employment of energetic *derivatives to the skin* over the spinal column, is permissible and serviceable. The results obtained by their employment in other affections, as well as in a few cases of myelitis, have been very encouraging, but at the same time the danger of producing ulcers, and of favoring the formation of bed-sores, and the great annoyance they cause the patients, are apt to deter physicians from using them. I believe that, when properly used, they can do no harm and often do good. Those which act rapidly and energetically should be chosen—the hot iron, or, in less serious cases, the blister. These counter-irritants should never be applied to places where the skin is already very anæsthetic; all parts that are exposed to continuous pressure should also be avoided. In severe and threatening cases (particularly in cases of ascending central myelitis) we need not hesitate to give them a very thorough trial, since the great danger in which the patients are placed justifies the use of severe and even dangerous remedies. Here the greatest advantage will be derived from two bold streaks drawn with ferrum candens, or from punctiform cauterizations on both sides of the spine, repeated every day or two. The favorable termination of Levy's case, in which the diagnosis of acute myelitis was in all probability correct, speaks highly in favor of this method.

The antiphlogistic treatment must, of course, be modified according to the age, constitution and general condition of the patients. The more robust the individual, the more energetic should be the antiphlogosis; in plethoric patients a moderate venesection may even be advisable.

The *galvanic current* should never be employed in the acute forms and the acute stage of myelitis. It is only in the treatment of the more chronic forms, and of the residua and sequelæ of the affection, that is permissible and proves valuable.

The cases reported by Lewin[1] and Hitzig[2] might possibly be adduced in opposition to this statement. In Lewin's case, however, the diagnosis was not quite certain. Under the galvanic treatment, which was begun on the twentieth day, the patient did remarkably well. In Hitzig's case, the chief affection was a subacute meningitis.

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[1] Deutsche Klinik. 1875. No. 11.

[2] Virch. Arch. Band XL. p. 445. 1867.

In addition to these therapeutic measures the most careful attention must also be paid, in the first stages, to the *feeding and the nursing* of the patient; his strength must be kept up, and all the evil consequences of the disease guarded against and combated. In this connection the following points must be attended to: absolute rest as far as possible, and of course in bed; the patient should lie often on his side or abdomen, and not exclusively on his back. It would hardly be possible to enforce the exclusively abdominal position, recommended by Brown-Séquard. Easily digested but nourishing food; no spirits, no tea or coffee; absolute mental quiet; avoidance of all violent movements of the body, especially of those caused by driving; careful attention to the skin, which must be washed regularly. In the milder cases a lukewarm bath may be administered. Above all things, every precaution must be taken to prevent the development of cystitis and decubitus; the directions for this will be found in the general part (Part I., p. 193).

If we succeed in tiding the patient over the first few weeks, or the acute stage of the disease, the case may be safely left for a time to nature, on which our chief reliance must be placed under all circumstances. Of course, the directions given for the diet and nursing must still be strictly followed out. We can, to a certain extent, count on the development of a reparative action in the organism, which will lead to at least a partial adjustment of the derangements. In all such cases we have to deal with subacute and chronic forms of myelitis, and the treatment of these forms, which is given at length in the succeeding chapter, then comes into play.

The time has now come for a more *supporting and stimulating treatment* (quinine, iron, good food, wine, cod-liver oil, fresh air, etc.), which will further the restoration and regeneration of the tissue; also for the employment of *baths* (thermal and brine baths), or of a mild *course of hydropathy;* particularly, however, for the employment of the *galvanic current.* For detailed information concerning the indications for these measures and the methods of employing them, the reader is referred to the section on the treatment of chronic myelitis.

Finally, specific internal remedies, such as nitrate of silver, chloride of gold and sodium, iodide of potassium, etc., are also indicated now. Strychnine may also be cautiously tried.

If the case be progressively improving, the patient may gradually be allowed to move about cautiously. He should be put through a course of chamber gymnastics, etc., before he is allowed to go about freely.

Finally, convalescence in the cases which run a favorable course, may be hastened by residence in a mountainous region, or by cold-water treatment, or by sea-baths. The patients, however, must always observe great caution for a long time, and avoid every excess, every occasion of over-exertion and any exposure to cold.

The *indicatio symptomatica* may be furnished by the most different symptoms. The most important, as a rule, is the careful treatment of the cystitis and decubitus, the rules for which have been given in the general part (Part I., p. 193). The relief of the pains and of the muscular twitchings and contractures that follow in their train, is also often very desirable. (Morphine, bromide of potassium, chloral, etc.) Sleeplessness may also require treatment. Against the asphyctic condition which sets in *sub finem*, and the impending cardiac paralysis, the usual irritant remedies are almost powerless. The anæsthesias, paralyses, atrophies, etc., which persist after the cessation of the disease, are most satisfactorily treated by electricity.

B. Chronic Inflammation of the Spinal Cord.—Myelitis Chronica.—Sclerosis. Gray Degeneration.

Definition.—We enter here on a domain, the limits of which it is exceedingly difficult to mark off. For the present we must include under the head of chronic myelitis, many affections that will doubtless be sifted out and more sharply defined in the course of time.

We understand by chronic myelitis *all those slowly developing processes in the spinal cord which run a tedious, lingering course without fever, and which in the present state of our knowledge are ascribed to chronic inflammation.* It comprises consequently the processes which are designated as chronic inflammation in its narrowest sense, as induration, as sclerosis and as gray degeneration; also a portion of the slowly developing processes of softening, and perhaps some of the cases in which cavities form in the cord.

We include here consequently forms of disease which vary very greatly in location and extent. In this chapter, however, we will consider only those forms which do not possess an exact localization, but are characterized rather by irregularity in seat and extent. The remaining forms, especially the funicular scleroses, etc., will be made the subjects of special chapters.

Etiology and Pathogenesis.

The question whether an increased *predisposition* to myelitis exists in certain persons, has not yet been sufficiently investigated, but it is at any rate exceedingly probable that it does. Here too, the hereditary neuropathic constitution presumably plays a very prominent role; numerous facts in the etiology of tabes, multiple sclerosis, etc., indicate this. Mental and corporeal over-exertion, dissipation, sexual excesses, syphilis, emotional excitement, care, grief, etc., can probably also act as predisposing causes. The disease is most common during youth and middle age, and in the male sex.

Chronic myelitis can, as we have already seen, proceed from the acute form. This is not an uncommon occurrence. Hence, all the causes of the acute must also be regarded as more remote causes of the chronic affection. The latter, however, can also be primarily produced by these same causes. The differences in the degree of the external injurious influences, or of the predisposition to the disease, which determine the occurrence of an acute affection in one case, and of a chronic affection in another, are still unknown. Of the exciting causes the following may be briefly mentioned: *catching cold*, which is one of the most important, and certainly one of the most frequent causes; often-repeated exposure, long sojourn in damp, cold localities, sleeping on damp earth, etc., are especially dangerous. *Bodily over-*

exertion, especially when combined with exposure to cold; hence the great frequency of chronic myelitis after military campaigns. Simple *concussion* of the cord without direct injury has already been mentioned (p. 346) as a possible cause of chronic myelitis; also *gradual compression* of the cord.

Whether *alterations in the circulation* within the cord, such as possibly follow suppression of habitual hemorrhages (the menses, bleeding piles, etc.), can be the starting-point of chronic myelitis, is still doubtful. The question whether the myelitis, which develops in *persons suffering from hemorrhoids*, has a causal connection with the latter and the disorders excited by them, or whether we have to deal here with a purely accidental coincidence, which, in view of the commonness of piles, would not be at all astonishing, must still be left open.

Sexual excesses are undoubtedly rather a predisposing than an exciting cause of chronic myelitis. Still, there are several cases on record, in which no other cause could be discovered to account for the disease. The same remark applies to continued, depressing mental emotions.

Syphilis is unquestionably a very fruitful source of chronic myelitis. Putting out of question the syphilitic affections of the vertebral column and the spinal membranes, and the specific neoplasms in the cord itself, which excite myelitis only indirectly by means of the compression they exert, the occurrence of subacute and chronic myelitis in syphilitic individuals or in persons who had previously suffered from the disease, is so exceedingly frequent, that the possibility of an etiological connection between lues and chronic myelitis cannot be denied. During the last few years I have myself met with at least a dozen such cases, some of which I dissected. It is true, that it is still impossible to decide, whether in these cases we have to deal with really specific myelitis, or with simple myelitis in persons predisposed by the syphilis to chronic inflammations. Judging, however, from the cases in which I had opportunities to hold autopsies, an account of which will be given elsewhere by Dr. F. Schultze, I must for the present regard the latter as the more probable. More attention should in future be paid to this question, which is therapeutically exceedingly important.

Chronic myelitis is occasionally developed as a sequel of various acute and chronic diseases. Langhans found chronic myelitis with softening and the formation of cavities, in connection with lepra. Chronic alcoholism, chronic lead-poisoning, and perhaps also chronic poisoning with other metals, can likewise lead to chronic myelitis.

Finally, its development from *irritation and diseases of peripheral organs* must be mentioned. Most of the cases of so-called reflex paraplegia belong in the domain of subacute and chronic myelitis. It is unnecessary to add anything here, to what has been already said in different parts of this work concerning the pathogenesis of this form of disease. We will only allude to a case recently published by Laveran, which illustrates well the difficulty of explaining the etiological connection between the affection of the bladder and the disease of the cord.

Pathological Anatomy.

In many cases of chronic myelitis, the *macroscopic* examination reveals no noticeable anomalies. The investigations of recent years have left no room to doubt, that very marked and widespread structural changes may exist in cases where the most careful macroscopic examination discloses absolutely no alterations either in the color or the markings, or even in the consistency of the cord. The changes are revealed, however, when hardened preparations of the cord are subjected to a microscopical examination. It is unnecessary to dilate on the suspicion, which this discovery throws on the statements of many excellent observers, who were content to declare a cord to be "normal" after a mere macroscopic or, at most, a hasty microscopic examination. There can be no reason to question the truth of the statement, that it is *impossible* to decide positively from macroscopic observation alone, or from a superficial microscopic examination of fresh specimens, that the cord is normal in structure.

Not unfrequently, however, chronic myelitis causes very considerable macroscopic changes. These affect, on the one hand, the *consistency* and, on the other hand, the *color* of the cord, or of circumscribed portions of it. There is almost always an increase in the consistency—a *hardening* or *sclerosis* of the cord; its substance is denser and firmer, and resembles coagulated albumen, or it may be still denser and difficult to cut, presenting a smooth section. This hardening is usually accompanied by a more or less pronounced gray, or yellowish gray discoloration of the tissue—hence the much-used name, *gray degeneration.*

In a minority of the cases only, do we meet with more or less extensive *softening.* This is observed rather in the subacute cases, or in the earlier stages of a chronic myelitis, which has developed from the acute form. It is sometimes met with, however, even in primarily chronic cases, *e. g.*, in the one reported by Keen. Sometimes we meet with more or less extensive and numerous cavities, which, in a number of the cases at least, are by several authors correctly ascribed to chronic myelitis.

The spinal cord in these cases rarely presents any considerable change in volume. An increase in volume, or swelling of the whole or of circumscribed parts of the cord, is least frequent; on the other hand, a slight diminution in volume, or atrophy, is very common. What was formerly called consumption of the cord, atrophy of the cord, and tabes dorsalis, in the anatomical sense of the term, are really nothing more than different forms of chronic myelitis. In such cases the cord is atrophic and thinner than usual; sometimes it is greatly shrunken. This change may be general and equable, so that the cord still presents the appearance of a cylindrical structure, but it is usually limited to its lower half; or the shrinkage may be in the antero-posterior direction, so that the organ has a more flattened, band-like appearance; or, finally, the surface is somewhat depressed only in isolated, circumscribed spots, or perhaps along the entire length of certain columns. Everywhere the gray, degenerated tissue shimmers through the pia.

The *localization and the area* of the chronic inflammation in the spinal cord may vary greatly. There may be merely isolated foci of disease, varying in number and size, and imbedded in healthy tissue. They are dense, reddish-gray or gray, often possess a gelatinous translucency, and are either sharply defined, or gradually shade off into the healthy tissue. When there is only one focus, which does not take in an entire cross-section of the cord, we speak of a *myelitis circumscripta.* When, as not unfrequently happens, there are several of these foci scattered irregularly through the cord, we have the *myelitis chronica disseminata*, or *sclerosis multiplex seu disseminata*, which is usually combined with an analogous, disseminated sclerosis in the brain.

When the entire thickness of the cord is involved, the disease, however, being more or less limited in a vertical direction, we have before us the very common form to which the name *myelitis transversa* has been applied. A portion of the cord of variable length, but usually not more than a few inches in span, appears grayish, translucent, shrunken and atrophied; the line of demarcation from the surrounding tissue is not very sharply defined, especially when, as is usually the case, secondary degeneration has set in. On section, the tissue appears more or less firm, dense and dry, or occasionally more succulent and soft.

In other cases the lesion involves only small and as a rule distinctly circumscribed portions of the cross-section of the cord, but extends farther in a vertical direction, sometimes stretching throughout almost the entire length of the organ. In this last case we have to deal with the so-called *band-shaped sclerosis*, or funicular degeneration. In this form of chronic myelitis, different parts of the cross-section may be involved. The affection may be limited to the gray substance, and may surround the central canal in the form of a cylinder of varying thickness; in this situation it not unfrequently leads to the formation of cavities—this is the so-called *periependymal sclerosis*, which Hallopeau has described minutely. Or it may be confined to the anterior horns of the gray matter, one or both of which may be affected to a varying extent longitudinally: this is the *poliomyelitis anterior chronica* (Kussmaul). Or it may be located exclusively in the white columns, usually involving a large portion of their longitudinal section, and almost always affecting both sides symmetrically. In this category belongs the much spoken of *gray degeneration of the posterior columns;* the degeneration may involve the entire thickness of these columns, or it may be limited to the funiculi graciles. The *sclerosis of the lateral columns*, especially of their posterior portions, and the sclerosis of the inner portion of the anterior columns, also belong here. We will speak more fully of these forms in another place. Finally, the chronic myelitis may also be confined to the periphery of the cord, forming a ring-shaped zone lying immediately beneath the pia, and corresponding to the normal cortical layer. This form is usually combined with meningitis. It constitutes the *ring-shaped, peripheral sclerosis*, or the *sclerose corticale annulaire* of Vulpian.

When the chronic myelitis extends over the greater part of the cord, it is termed *myelitis chronica diffusa*. This extension, however, is usually not uniform, some portions being much more intensely affected than others. In many cases the myelitis is at first circumscribed, but becomes more or less diffused in course of time.

A close examination reveals in very many cases the presence of the *secondary degenerations*, which have been often alluded to already, and which will be more fully described in another chapter. In an upward direction, it is chiefly the ascending degeneration of the posterior columns, reaching to the medulla oblongata, which attracts the attention of the observer—in a downward direction, the descending degeneration of the posterior portions of the lateral columns, reaching to the end of the lumbar medulla. The transition from these degenerated parts to the actual myelitic centres, is usually a very gradual one.

The *meninges*, as a rule, take part in the chronic myelitic process. They also present the signs of chronic inflammation—cloudiness, thickening, adhesions, calcifications, etc. It is unnecessary to describe these changes here.

The *nerve-roots* are in many cases gray, translucent, exceedingly atrophic, and converted into a wavy connective tissue, rich in fat, but containing few or no nerve-fibres.

The *peripheral nerves* also are not unfrequently found in a state of degenerative atrophy. The *muscles* are then in the same condition; they appear very atrophic, pale, rich in connective tissue, and occasionally very abundantly supplied with adipose tissue. These points will be considered more in detail, when we come to speak of the forms of chronic myelitis, that are most liable to be followed by these degenerative processes.

Furthermore, at the autopsy we often find changes in the *joints* (erosion, deformity or destruction of the cartilages, thickening of the capsule, serous accumulations, etc.); more or less extensive and often gangrenous *bed-sores* on different parts of the body; marked *vesical changes* (hypertrophy and ulceration or diphtheritis of the mucous membrane, hypertrophy of the muscular coat, etc.) with consecutive pyelitis and nephritis, etc. We find also many other changes which are partly consecutive to the severe, chronic spinal paralysis, partly symptomatic of accidental, intercurrent diseases, but which it is unnecessary to describe here.

We possess a great abundance of isolated observations bearing on the *microscopical lesions* of chronic myelitis. In general, however, there is still but little unanimity on the subject, least of all concerning the interpretation to be placed on what has been discovered. We cannot, of course, enter here into all the details, since many of the points in dispute are as yet undeveloped, and for the practising physician of but little importance. Neither have we to speak of the methods of investigation, as that is a subject which belongs in the domain of pathological anatomy; essentially, all the discoveries have been made by the study of hardened preparations, colored and rendered transparent by various methods.

It is probable that many different processes are still classed together under the head of chronic myelitis. The changes enumerated in the following paragraphs are, however, common to all these forms, and constitute in all probability the essence of the process.

Changes in the connective tissue or the neuroglia: increase in the thickness and width of the individual fibres and septa of the reticulum; gradual transformation of the tissue into a delicately fibrous, fibrillated connective tissue. According to Frommann the fibrillæ are developed within the old, enlarged neuroglia fibres and cells. They form finally a dense tissue, composed of delicate, fibrillated fibres, usually arranged in long wavy bundles. The neuroglia-cells enlarge, and their nuclei undergo proliferation; they become more numerous and more distinct. Nowhere can Deiter's cells be found more perfect and more developed than in chronic myelitis: truly gigantic specimens with numerous processes are often found. The change consists consequently in a gradual conversion of the normal neuroglia tissue into a fibrillated, delicately fibrous connective tissue, accompanied by proliferation of the cells and their nuclei.

Changes in the nervous elements: sometimes the *nerve-fibres* are swollen, the medullary sheath being irregularly thickened, crumbly and in a state of commencing degeneration; in this case numerous swollen *axis-cylinders*, which present spindle-shaped enlargements and are fused with the medullary sheaths, are also found. More frequently, however, we find pronounced atrophy or a starved condition of the fibres, with destruction of the medullary sheath, sometimes by fatty degeneration, sometimes without apparent change of this sort. The naked axis-cylinders then often remain in *statu quo* for a considerable time; but finally, they too undergo atrophy and become shrunken and destroyed. Sometimes they are found stiff, brittle, very brilliant, and indurated.

The *ganglion-cells* are sometimes clouded and swollen, and contain vacuoles. More frequently, however, they are diminished in size, strongly pigmented, shrunken, atrophied and indurated, and are finally converted into small, unrecognizable, angular structures, in which neither nucleus nor nucleolus can any longer be discovered, and which have lost their processes.

Changes in the vessels.—The walls of the small arteries and veins, and of the capillaries as well, are considerably thickened, and their calibre is lessened. This of course varies very greatly in degree in different cases. The walls, especially the adventitia, are converted into a thick, homogeneous, often indurated mass, presenting proliferation of nuclei, collections of fat- and pigment-granules, and not unfrequently also granule-cells. The perivascular space is filled with cells and exudation; fusion of the wall of the vessel with the surrounding, indurated connective tissue; sometimes aneurysmal dilatations of the vessels.

Appearance of heterogeneous elements: of these the fat-granule-cells (Fettkörnchenzellen) are the most frequently met with;

they are sometimes numerous, sometimes scanty, at one time deposited around and in the walls of the vessels, at another scattered through the connective-tissue septa, and again arranged in rows, apparently in the spaces that, under normal circumstances, contain the nerve-fibres. This very difference in distribution indicates different modes of production.

Corpora amylacea are also usually found; they are sometimes present in immense numbers, scattered throughout the entire tissue, but at other times only a few can be discovered.

All these things will be found in the different stages of chronic myelitis. Not unfrequently they will all be found at the same time in the same cord, provided different parts of it be the seat of different stages of the process.

It is evident, that the different age of the process must occasion characteristic differences in the histological picture. All the details of these differences have not yet been thoroughly settled, but still the various stages of the disease can be characterized histologically about as follows:

1. *The earliest stage.*—Thickening and swelling of the interstitial tissue; greater succulence of the tissue; proliferation of the nuclei, slight swelling of Deiter's cells; commencing thickening and alteration of the walls of the vessels; nerve-fibres rather swollen, medullary sheath in a state of incipient degeneration; axis-cylinders often no longer distinct; ganglion-cells clouded, irregularly swollen, with fewer processes, etc.; more or less abundant fat-granule cells, and a few corpora amylacea.

2. *Later stage.*—More marked increase of the interstitial tissue, close network of thick neuroglia-fibres, proliferation of the nuclei, increase of the neuroglia-cells, great thickening of the walls of the vessels; distinct atrophy of the nerve-fibres, a number of which have disappeared. On the other hand, the axis-cylinders sometimes survive for a very long time (particularly in the so-called multiple sclerosis, for which it seems to be to some extent characteristic.—Charcot, Leyden). Ganglion-cells atrophic, sclerosed and in part destroyed; fewer fat-granule cells, more corpora amylacea. The tissue in general, of greater density.

3. *Last stage.*—Almost exclusively connective tissue; a dense

tissue, composed of delicate fibrillated fibres in a state of inextricable entanglement, with tolerably numerous nuclei and cells; the latter often strikingly developed and distinct. Walls of the vessels greatly thickened, vascular lumen narrowed or in rare instances enlarged. Only a few, isolated nerve-fibres are still recognizable; the greater part of the fibres have disappeared, and most of those which remain are atrophic; many naked axis-cylinders. Ganglion-cells shrunken to unrecognizable elements, or entirely destroyed. Very few fat-granule cells, but corpora amylacea, on the other hand, usually very numerous. The entire tissue very dense, firm, dry and shrunken. These are the appearances presented in the oldest cases, which have lasted several or many years.

In many cases extensive cavities are found in the cord—this is probably the terminal stage of a very chronic myelitis characterized by softening. We shall return to these cavities in another chapter.

These are the chief lesions found in chronic myelitis. When we attempt to interpret them, various questions force themselves on our notice, the solution of which still presents considerable difficulties. We cannot, however, well avoid devoting a brief space to a consideration of them.

In the first place, the question arises, *whether in all these processes, we have really to deal with a chronic inflammation?* We cannot spare time for a close examination of this general pathological question. According to the principles that are still generally accepted, the term chronic inflammation (cirrhosis, etc.) is in accord with the anatomical lesions, which are analogous to those found in other organs when the seat of chronic inflammation. The matter, however, is still very open to discussion, especially the question whether the process in many cases may not be one of simple or of degenerative atrophy. This interpretation may apply to the so-called "secondary degenerations," which some authors are disposed to class among the chronic inflammatory processes (Charcot, Hallopeau). The anatomical picture of secondary degeneration can certainly resemble very closely that of chronic inflammation. Joffroy asserts that in experimental myelitis, he found swelling and hypertrophy of the axis-cylinders in the actual focus of inflammation, but not in the parts which had undergone secondary degeneration; this would indicate a difference in the two processes. This observation, however, requires confirmation, especially as it contradicts the statements of other observers. In secondary degeneration we have to deal essentially with that form of degenerative atrophy which occurs in the nerve-trunks, when they are cut off from their nutritive centres. (Compare our description and explanation of this in Vol. XI., p. 412.) It is true that this atrophy also is not unfrequently spoken of as an actual inflammation; it must then be regarded, however, as a specifically neurotic inflammation. Very plausible also is the theory, that in such cases there is a primary degeneration of the nerve-fibres, the products of which exert an irritant action on the interstitial tissue, and excite in it a slow, chronic inflammation (sclerosis). A process of this sort might well occur primarily in many forms of the band-shaped affection, and it would furnish a simple explanation for the fact, that interstitial, chronic inflammatory changes exist also in the gray degeneration or degenerative atrophy of the spinal cord.

This brings us to a second very important question, namely, whether the chronic inflammatory process always has the same point of departure, be this from the connective tissue or from the nervous elements? Whether, in other words, *we must assume an interstitial or a parenchymatous inflammation, or perhaps both together?* To this question also we are as yet unable to give a decided answer. It seems both possible and probable, however, that both forms of inflammation may occur; authorities whose opinions carry weight, maintain this and claim that they possess observations which prove it. In general, however, we have to deal here with assumption against assumption. Charcot and his disciples do not hesitate to distinguish the *interstitial* myelitis or sclerosis from the *parenchymatous* form. In the former it is claimed that the process begins in the connective tissue, the nerve-fibres not being involved until the affection has reached its later stages; hence the fibres and axis-cylinders remain unaltered for a long time (disseminated sclerosis). In the latter, on the contrary, the process begins with irritation and degenerative atrophy of the nerve-fibres and ganglion-cells, and involves secondarily the interstitial tissue (band-shaped sclerosis of the posterior columns, progressive muscular atrophy, probably also secondary degenerations). It is, in the present state of our knowledge, difficult to demonstrate the correctness of this view. An unprejudiced examination of preparations from both forms of the disease shows that there are no essential differences in the histological appearances, that the same alterations may occur in both forms, and that all theories concerning the mode of their production are more or less gratuitous.

Theoretically the thing is certainly possible and even probable, but it can by no means be admitted as proven. It is, in fact, very difficult to decide the question from a purely anatomical standpoint. At all events, it does not seem to us justifiable to confine the term chronic myelitis solely to the so-called interstitial sclerosis, and to deny the character of an inflammatory process to the so-called parenchymatous sclerosis. To us, at least, it seems better and simpler to designate for the present all these forms together as scleroses, and to differentiate them chiefly according to their localizations.

Another and a better differentiation will only be possible when a third question is decided, namely, whether *the pathological process in all these forms of disease, which histologically present essentially the same picture,* is actually the same? Whether, consequently, the different conditions described as induration, gray degeneration, multiple sclerosis, secondary degeneration, etc., are to be ascribed to one and the same distinct disease, namely, chronic myelitis, of which they constitute only different forms, or whether they represent essentially different diseases? In our opinion it is still impossible to decide this question with positive certainty. The French school in particular classes the secondary degenerations with myelitis and the band-shaped scleroses. With all reverence for the value of the evidence furnished by the histological appearances, we cannot repress a modest doubt of the correctness of this view, a doubt which is based chiefly on physiological and clinical considerations. There is another point on which, as it seems to me, most observers agree, and that is that the processes in tabes and multiple sclerosis are not entirely identical, but present certain differences which cannot as yet be defined with satisfactory precision.

There undoubtedly remains much to be done in this department, but judging from the great strides that have of late been made in the development of the pathological histology of the spinal cord, we may confidently expect that the conception of chronic myelitis will be cleared from its obscurities in a not very distant future.

Pathology of Chronic Myelitis.

Symptoms.

In consequence of the great diversity of the processes and forms of disease, that are included under the term chronic myelitis, it is difficult, if not impossible, to draw in connected strokes a *general clinical picture* of the affection.

The following description consequently applies more to the general type of chronic myelitis, and particularly to the so uncommonly frequent myelitis transversa, where one large focus of disease exists at any height in the cord, or where there are several foci, one of which, however, determines chiefly the clinical picture.

In the majority of the cases belonging in this class, there is a slow, creeping, *almost unnoticed development of spinal symptoms*, which vary in nature and location. *Sensory disturbances* are usually the first to attract the attention of the patient, but they ordinarily discommode him but little. They consist of abnormal sensations in the lower, more rarely in the upper extremities, paræsthesiæ, anæsthesiæ, and the like, which, however, are subject to frequent fluctuations, and may even at times entirely disappear. They are frequently accompanied by a girdle-sensation in the trunk at a varying height, or perhaps also in the extremities. Severe painful sensations are rare, as are also hyperæsthesiæ of any degree of severity. All these symptoms are exceedingly variable and inconstant, and only very gradually attain any great degree of intensity and stability.

Slight *motor disturbances* next present themselves and develop in a very analogous manner. They may, however, open the scene. A feeling of weakness and of slight fatigue, and a certain heaviness and uncertainty of gait, are the first symptoms to attract notice. The patients are incapable of making any great exertion, and very quickly become fatigued. These symptoms also may fluctuate considerably in intensity; they are often neglected for a long time and ascribed to all possible causes but the right one, until finally, in common with the sensory disturbances, they assume graver significance.

Sometimes the disease commences with *vesical weakness*, with slight incontinence or retention, more rarely with pronounced paralysis of the bladder. These symptoms may exist for weeks or months, before the other signs of the disease make their appearance. *Constipation* is almost always present, even in the earliest stages of the disease.

After a time, as the disease progresses, all the above-mentioned symptoms are found in combination; a slow, progressive, or sometimes an intermittent *aggravation* of the symptoms is noticed. Not unfrequently we observe a more rapid advance of the disease, a sudden, marked aggravation of the symptoms, under the influence of some injurious agency, such as over-exertion, exposure to cold, sexual excesses, the improper use of baths, and the like. Finally, after the disease has lasted a variable time—several months or even years—the clinical picture is thoroughly developed, usually taking the form of typical spinal paraplegia with all its consequences. There is distinct *paralysis*, varying in degree from paresis to complete paralysis; it generally takes the form of paraplegia, but may assume any other possible form. *Anæsthesia* of greater or less intensity is rarely wanting. It varies in point of extension, but usually involves the lower extremities and the trunk up to a variable height. Partial paralysis of sensation and retardation of the sensory conduction occur. Paræsthesiæ and dysæsthesiæ in the paralyzed parts are not rare. Usually, however, the motor is more intense than the sensory paralysis. The *sphincters* are almost always involved; the different varieties and degrees of vesical paralysis are met with, and similarly there may be mere weakness of the sphincter ani, or involuntary and unconscious passages from the bowels. A marked *increase of the reflex activity* usually occurs; a spontaneous *twitching of the muscles,* which is often accompanied by severe pains, and muscular contractions and contractures, etc., are also met with. The sexual power is wont to expire early.

In the form of chronic myelitis, to which the above description chiefly applies, the *reflex excitability may be lost* after a time. *Progressive atrophy of the muscles,* changes in the skin, nails and joints, and *chronic decubitus,* with all its consequences, may also occur. When vesical derangements exist, *cystitis* rarely remains long absent. All these symptoms, however, are, as a rule, developed very slowly, and only very gradually attain dangerous intensity and extent.

The disease may terminate in different ways. A more or less complete recovery may very slowly take place, or the affection may become stationary and remain so for many years, or a fatal termination may be gradually brought about in various ways.

The clinical history is essentially the same as that of the analogous form of acute myelitis, the only differences being the very slow and gradual development of the disease, the entire absence of fever, and the usually excellent state of the general health of the patient. The duration of the disease is always very protracted; it must be reckoned by years.

The preceding clinical picture applies, as has been already stated, only to one of the many forms of chronic myelitis. It would require too much space, to describe in a similar manner the other forms, in which the seat of the disease is different, and we must content ourselves for the present with the brief characterization of them which is given below. For further particulars concerning them, we must refer to the appropriate chapters.

When we *analyze the individual symptoms* of chronic myelitis, we meet with the same pathogenetic conditions as in the acute disease. A detailed description and minute investigation of them would consequently only lead to needless repetitions. Still, there are many points, which it is necessary for us to examine more closely before proceeding further.

Among the *sensory disturbances* the *symptoms of irritation* play a comparatively unimportant rôle. There are cases in which pain is entirely absent throughout the entire course of the disease. In other cases, however, very violent pains are experienced, particularly during the stage of development, or very severe paroxysms of pain occur at intervals during the entire course of the disease. The spasmodic muscular contractions of the later stages are usually accompanied by very intense pains, and I have seen cases in which peripheral sensory irritations, such as the dressing of a bed-sore, passing the catheter, etc., excited violent radiating pains in the back or the extremities. The peculiar, vibrating, painful sensation produced by touching the skin, to which Charcot has applied the term dysæsthesia, is also met with.

The so-called *paræsthesiæ* are more frequently observed: the girdle-sensation, formication, numbness, cutaneous burning, furry feeling, etc., are very common. Finally, *anæsthesia* is almost regularly developed in the later stages of the disease; there are cases, however, in which it is entirely or almost entirely absent. The anæsthesia may vary greatly in degree. All possible forms of sensory paralysis, from mere slowness of conduction to the most complete anæsthesia, may occur. It is not necessary to describe these things in detail; for an explanation of them we may refer to the general part (p. 66 et seq.).

Among the *motor disturbances* also, the *symptoms of irritation* are of subordinate importance, particularly in the first stages. Usually there is only an occasional, slight, muscular twitch, or a sudden twitching movement of the legs, or the like; there may also be slight muscular contractions and trembling of the legs after fatiguing exertion, or when the weight is laid on the toes.

The *signs of paralysis*, however, are usually much more important and more prominent. Ordinarily, the first symptoms are marked weakness, loss of tone, and a sense of fatigue. In the early part of the disease these symptoms are not unfrequently relieved by moderate movement, such as walking; the patients feel stiffer and more fatigued at starting out than after they have walked for a time. As the disease progresses the stiffness and helplessness become more marked and are objectively evident. The movements are slow and stiff; the fingers and toes, for instance, can no longer be moved about quickly, or this is only possible with the help of many associated movements. The legs feel heavy, "as if they were filled with lead," and the feet are dragged along the ground, the toes readily catching in every slight inequality. The walk has a pronounced paralytic character.

Gradually both walking and standing become impossible. When the patients attempt to stand they collapse; their legs can no longer sustain them. Even while lying down the movements become progressively weaker and less frequent, until finally, the case terminates in complete paralysis. This may vary greatly in form. In the great majority of the cases it takes the form of paraplegia, but it may occasionally appear as hemiparaplegia or spinal hemiplegia, or even cervical paraplegia, etc.

The *spastic symptoms* may return again in a later stage. First of all, a gradually increasing contraction of the muscles sets in; this is manifest at first only during passive motion, but it soon becomes evident also during the voluntary movements, which are impeded and retarded by it. The characteristic "spastic" walk, which we have described elsewhere (Part I., p. 97), is now developed. The muscles become more and more tense and rigid, until finally a state of permanent and more or less marked contracture is produced. This may affect either the extensors or the flexors, the latter being, as a rule, attacked only in the later stages of the disease. The adductors are likewise almost always involved. The stiff and paralyzed limbs are not unfrequently shaken by twitches and spasmodic movements, which either arise spontaneously or are excited by sensory irritations. These twitches and movements are often exceedingly active and may resemble voluntary movements; they are often accompanied by severe pains. They are most readily excited by sensory irritations: cutaneous irritation, catheterization, micturition or defecation, traction on the tendons, etc. They may develop into long-continued, convulsive tremor of the lower extremities. Sometimes all voluntary movements are accompanied and followed by this spasmodic muscular action.

The various, special disturbances of motion, such as the ataxia, the tremor accompanying voluntary movements, etc., which occur in other distinctly localized forms of chronic myelitis, will be described more minutely in the appropriate chapters.

All the motor disturbances are very readily explained by the chronic inflammatory processes going on in the motor conducting tracks and central apparatus, although it is not always possible to determine the exact point in the central nervous system, at which these processes are located. Of late it has become customary to ascribe the spastic symptoms, which develop during the later stages of the affection, to secondary degeneration of the lateral columns. It cannot be denied, that there are good grounds for this assumption.

The *vaso-motor disturbances* in chronic myelitis are, as a rule, not of a very striking nature. The coldness of the feet, which is very frequently complained of and proves very annoying to the patients, probably belongs here, as does also the often observed, cyanotic, bluish-red color of the lower limbs. In consequence probably of the very slow development of the obstruction to conduction, the vascular innervation is able to adapt itself to the loss of a part of its spinal centres, so that the resulting derangements are not very marked.

The *state of the reflex irritability* varies. In the majority of the cases it is increased and is very active. Very curious things are often observed in this connection; reflex movements of the strangest kinds are produced by irritation of all imaginable sensitive regions. Tickling the soles of the feet causes active jerking and kicking movements in the paralyzed legs; colic pains, the introduction of a catheter, the act of dressing a bed-sore, etc., may excite movements in the legs. I have seen evacuation of the urine produced by irritating the skin of the feet, or by introducing the finger into the rectum; erections produced by rubbing gray ointment into the thighs; a discharge of fæces occur during the dressing of a bed-sore, etc. The tendon-reflexes are also, as a rule, abnormally active, often excessively so. The slightest irritation of the tendon of the quadriceps extensor femoris causes a reflex movement, which can readily be made to develop into clonic spasm. The most marked clonic spasm of the muscles of the calf can be excited by passive flexion of the foot; reflex movements can also be produced by irritation of the tendons of the adductors and of numerous other muscles.

Both of the conditions which are known to lead to increase of the reflex activity—destruction of the connection between the reflex apparatus and the brain, and abnormal irritability of the gray substance—may be present in chronic transverse myelitis. It is probable that in most of the cases the increase is due to a combination of these two conditions.

On the other hand, there are cases in which the reflex irritability is diminished, or even entirely abolished. In such cases we must, in accordance with the views at present held, assume either that the gray substance is very extensively involved, or that the conducting power of the nerve-roots which come into play, is destroyed by the myelitis.

There is an unmistakable parallelism between the state of the reflex excitability and the state of the *electric irritability of the nerves and muscles.* When the reflexes persist, the electric excitability is also, as a rule, preserved. In the majority of the cases, I have found the latter to be both qualitatively and quantitatively normal. In a few isolated cases there was a distinct increase in the faradic and galvanic excitability (without qualitative anomalies) of the nerves of the paralyzed limbs; in other cases there was an unimportant quantitative diminution of this excitability. I am as yet, however, unable to say, whether or not this pathological state will afford us any assistance in determining the seat, stage, or course of the disease.

In those cases, however, in which the reflexes are abolished in consequence of extensive implication of the gray substance, atrophy of the muscles usually sets in, accompanied by loss of their faradic excitability (probably with the reaction of degeneration in the majority of the cases). In certain forms of chronic myelitis, which we can with great probability locate in the anterior horns of the gray matter, this state of affairs constitutes the rule, the loss of electric irritability occurring remarkably early and rapidly.

Further and more accurate investigations of these phenomena in chronic myelitis are greatly to be desired.

There is a very close connection between the electric irritability and the *state of the nutrition of the muscles.* Usually the latter is intact and good, and it can remain so during the entire course of the disease. In other cases a slow, progressive atrophy of the muscles sets in in the later stages—a sign that the gray substance has become more extensively involved. In still other cases a rapidly progressive atrophy of the muscles is observed, which sets in early, and is accompanied by the reaction of degeneration. The territorial extent of the atrophy varies greatly; it depends on the extent of the central disease. Sometimes the upper extremities are chiefly or alone affected by the atrophy, the lower limbs presenting simple paralysis without atrophy. This is easily accounted for by differences in the condition of the gray substance of the cervical and the lumbar enlargements.

In regard also to other *trophic derangements,* the individual cases vary greatly. They are entirely wanting in some forms of chronic myelitis, of which we will presently give brief descriptions; as a rule, disorders of sensation and of the bladder are also wanting in such cases. Frequently, however, and more especially in cases of myelitis transversa, decubitus is developed sooner or later, generally in the chronic form already described (Part I., p. 122); sloughs form in the usual places of predilection, their formation being not unfrequently hastened by more or less accidental agencies (sitting or lying in the same position for a long time, unusually severe pressure or other mechanical injury, a long railroad journey, febrile diseases, etc.). Sometimes, when the patient has not been nursed with proper care, the attendants will be astonished by the unexpected discovery of a bed-sore, which has already attained large dimensions, on the nates or over the sacrum. The sore may develop and spread with more or less rapidity, but it usually runs a chronic course. When great attention is paid to cleanliness, etc., the parts first affected may, after separation of the sloughs, heal up in the course of a few weeks or months; fresh bed-sores, however, constantly form in other situations, often in considerable numbers, or the already cicatrized spots break out afresh on the slightest provocation. The patients, even when they are covered with large ulcers, may, if carefully nursed, drag out a miserable existence for a long time, until finally death ensues from exhaustion. Under all circumstances the formation of extensive sloughs is an unfavorable sign; it marks the beginning of the end. In some rare cases large decubitus ulcers may become permanently cicatrized, even though there be no noticeable improvement in the primary disease. I have at present one such case under observation. With respect to the pathogenesis of the bed-sores in chronic myelitis, we have nothing to add to the remarks made in the place mentioned above.

In the forms of chronic myelitis which more especially engage our attention here, the *sphincters* are almost always involved in the paralysis to a greater or less extent. The vesical functions in particular are almost regularly impaired at an early period; there are some exceptional cases, however, in which the bladder remains unaffected throughout the entire course of the disease. In many cases the vesical paralysis is the first symptom, and precedes the other manifestations of the disease often by a considerable period (Laveran's case). It may present any of the different forms and degrees which have been enumerated elsewhere (Part I., p. 132); it may vary from slight retention of urine to complete incontinence, or constant dribbling of urine. The variations in the localization and extent of the myelitis explain sufficiently these differences in the vesical symptoms. Naturally, cystitis with all its evil consequences, which render the nursing and treatment of the patient so much more difficult, does not remain long absent under such circumstances. Pyelitis and nephritis may set in later, and hasten the fatal termination. The functions of the rectum usually suffer in an analogous manner to those of the bladder.

Sexual power is usually impaired early. As a rule, it diminishes with more or less rapidity, and finally disappears entirely. Notable exceptions to this rule are met with, however, particularly in cases of incomplete paraplegia, and in those forms of chronic myelitis which affect chiefly or exclusively the voluntary motor apparatus.

The *general nutrition* often remains perfect for a very long time. It is not at all rare to meet with patients, whose blooming and well-nourished appearance presents a most striking contrast to the state of perfect helplessness, to which they are reduced by a complete paraplegia or some other form of paralysis. This is to some extent characteristic of certain special forms of chronic myelitis, which will presently be described. In all the more severe and progressive cases, however, a constantly increasing disturbance of the general nutrition is observed, though usually not until after the development of cystitis and bed-sores. Loss of appetite, fever, repeated chills, digestive derangements, progressive emaciation and finally complete exhaustion generally constitute the final symptoms in such cases, provided some intercurrent disease does not terminate life sooner.

With regard to *the disorders of the cerebral nerves and of the brain itself*, we have but little to say. There are cases of chronic myelitis, *e. g.*, of tabes and multiple sclerosis, in the clinical picture of which disorders of certain cerebral nerves, such as the optici, the nerves of the muscles of the eye, the faciales and the hypoglossi, occupy an important position; there are other cases, in which psychical disturbances and disorders of speech and of the voice are equally prominent. We will return to these symptoms in the appropriate chapters. In the cases of myelitis transversa, however, which interest us more especially at present, cerebral symptoms and disorders of the cerebral nerves are usually entirely wanting throughout the whole course of the disease, or, at most, they occur during the agony. Finally, there are cases in which the process slowly ascends until it at last reaches the medulla oblongata; disorders of deglutition, speech, respiration, circulation, etc., are then produced, and bring about the fatal termination. The sequence and grouping of these symptoms follow naturally from the functions of the cervical marrow and the oblongata, and it is unnecessary to describe them in detail, especially as they present very great differences in the individual cases.

Course—Duration—Terminations

When the chronic form of myelitis is developed from the acute form, it is preceded by the symptoms of the latter; some of these symptoms undergo a gradual retrogression and the general health improves, but the paralyses, pains, contractures, etc., persist and subsequently become slowly and gradually more developed.

In the other cases, we observe the slow, lingering development, which has been briefly described above. Of course the individual cases do not resemble each other exactly in every particular, but the fundamental characteristic of the disease always remains the same; it is the successive, gradual appearance of the various spinal symptoms, their development from insignificant beginnings which often attract little or no attention. Sometimes the development of the affection, instead of being continuous, is interrupted by intervals of more or less marked improvement; these alternate with exacerbations and slight relapses, each of which leaves its mark in an increase of the disturbances.

When the disease has attained a certain height it may remain stationary for many months or even years, or it may slowly progress and terminate in different ways. The great multiplicity of the morbid conditions included under the term chronic myelitis, makes it impossible to give even an approximate estimate of the *duration* of the disease. All we can say is that it is an affection whose duration can only be measured by years. In not a few cases, many years elapse before the final termination is reached, while in others the disease may persist during the lifetime of the patients, without directly threatening existence.

It very rarely terminates in *recovery*, and when it does the cure is usually exceedingly incomplete. Moreover, relapses are very liable to occur, and they furnish good reason to doubt the reality of the cure. Various residua, such as paralyses, atrophies, partial anæsthesias, deformities, vesical disorders, etc., often remain behind permanently, even when the actual morbid process has undoubtedly ceased; hence the recovery must be regarded as incomplete.

In one relatively mild case that came under my own observation, an intercurrent ileo-typhus exerted a very favorable influence on the course of the disease. In another case, an intercurrent scarlatina seemed to exert a similarly favorable influence.

A slow *progressive course*, leading gradually and in different ways to a *fatal termination*, is, however, so much more common as to be almost the rule. The process may slowly creep upwards, until, at last, disturbances of deglutition and respiration set in, and death is caused by asphyxia. Or the secondary affections caused by the myelitis, the decubitus, cystitis, pyæmic and septicæmic infection, etc., destroy the vital powers of the patient and lead to death by exhaustion. In such cases we sometimes observe a peculiar final stage, or stage of agony, in which death is preceded for days by high fever, chills, somnolence, delirium, signs of collapse, etc., for which the autopsy reveals no cause. Finally, death may be due to the development in the medulla oblongata or in the brain of a process analogous to the myelitis, or it may be caused by accidental, intercurrent diseases (pneumonia, typhus, choleraic diarrhœa, etc.).

Different Forms of Chronic Myelitis.

In what precedes we have sought to delineate the clinical history of *myelitis transversa;* if we exclude from consideration those well-marked forms, which are sharply localized in particular parts of the cord, and to which special chapters will be devoted, it is the most common and typical form of the affection known as chronic myelitis in its narrower sense. To briefly recapitulate, the typical picture of myelitis transversa is about as follows:

Slow development and persistence of spinal symptoms, which vary according to the height at which the disease in the cord is located; symptoms of irritation not very prominent; girdle-pains, pains in the back and paræsthesiæ, indicating the seat of the disease; signs of sensory and motor paralyses occurring together or in succession; early development of a more or less complete paraplegia, the upper limit of which can usually be readily determined by the disturbances of sensation and motion; weakness of the bladder and rectum; reflex irritability usually increased; general health good until the later stages, when it is impaired by the cystitis and bed-sores.

The symptoms vary somewhat, according to the location of the morbid process in the cord. When the *lumbar enlargement* is affected, we find complete paraplegia extending up to the hypogastric region, anæsthesia of equal extent, paralysis of the bladder and rectum, loss of reflex excitability, and usually muscular atrophy. Bed-sores develop early.

When the process is located in the *dorsal region* of the cord, paraplegia also exists, but the sensory and motor paralysis extends higher up—to the level of the nipples or even further; the muscles of the abdomen and back are involved. Expiration is impeded. Paralysis of the sphincters, bed-sores, etc., as in the preceding form; characteristic of the dorsal affection, however, are the often very marked increase of the reflex actions, the absence of muscular atrophy, and the appearance in the later stages of signs of irritation, contractures etc., in the muscles.

When the *cervical enlargement* is the seat of the disease, the usual initial symptoms are observed, but they affect principally the upper extremities, and the paralysis which follows frequently takes the form of paraplegia brachialis—the hands and arms are affected first and chiefly, the paralysis of the legs not appearing until later. When the affection is at its height, all the four extremities are more or less completely paralyzed. Paralysis of the sphincters, decubitus, and increase of the reflexes in the lower extremities, are present here as well as in myelitis of the dorsal region; the impeded inspiration, the oculo-pupillar symptoms, and the atrophy of the upper extremities with extinction of their reflex activity, are, however, characteristic of the cervical affection. In consequence of the loss of their reflex activity, the upper present a striking contrast to the lower limbs. The electric irritability is diminished or destroyed in the atrophied muscles.

When only the *upper part of the cervical marrow* is attacked, the clinical picture varies somewhat from the above. There is paralysis of all four extremities, it is true, but the nutrition of the upper limbs remains intact, and their reflex activity is rather increased than diminished. Moreover, severe disturbances of respiration (paralysis of the diaphragm), difficulty of swallowing, impairments of speech, vomiting, singultus, etc., occur which impart an exceedingly threatening character to the clinical picture.

A particularly common form of chronic myelitis is the so-called *compression-myelitis*, which occurs in connection with any disease, that occasions a slow compression of the cord. We have given a detailed account of this form of the affection in another place (p. 320), to which the inquirer is referred. We shall content ourselves here with the statements, that the clinical picture corresponds perfectly with that of spontaneous transverse myelitis, and that the only thing, which is at all distinctive and characteristic of a compression-myelitis, is the existence for a variable period preceding its development, of initial symptoms referable to some particular portion of the cord (severe pains and cramps, local paralysis, atrophy, or anæsthesia, dorsal pain and stiffness, local hyperæsthesia, etc.).

The form of chronic myelitis which is confined to the anterior horns of the gray matter, *poliomyelitis anterior chronica* (see Section No. 16) is, as a rule, readily recognized. It is characterized by a slowly increasing weakness and paralysis of the limbs, which advances progressively from below upwards; by distinct and sometimes rapid atrophy of the muscles, with loss of the faradic irritability (and probably with development of the reaction of degeneration); by the absence, or the very slight development of the sensory disturbances, which frequently exist only in the initial stage, in the form of dragging and tearing pains; also by the extinction of the reflexes, and the complete absence of decubitus and of vesical weakness. This is the affection which Duchenne has described under the name "Paralysie générale spinale antérieure subaiguë," the localization of which in the anterior gray substance is at all events exceedingly probable.

It is as yet impossible to give an accurate clinical description of that form of chronic myelitis, which is limited to the central gray substance, and to which the name *myelitis centralis* has been given. This form is the myélite periépendymaire of the French, and it probably includes also a portion of the cases of hydromyélie and syringomyélie. Many cases present absolutely no symptoms, while others, as it appears, present the clinical picture of progressive muscular atrophy, but with more prominent paralytic symptoms. Hallopeau ascribes to this form of the disease, circumscribed paralyses of different parts with atrophy and loss of electric irritability, vague pains, very little or absolutely no anæsthesia, sometimes tremor, and the like. The clinical picture is consequently so exceedingly vague and uncertain, that a positive diagnosis cannot be made from it.

In some cases the chronic myelitis gradually extends until it involves the entire cord; it may then be termed *myelitis universalis progressiva*. This form is characterized by progressive weakness and paralysis of the muscles, extending slowly from below upward, rarely in the reversed direction, while the muscular contractions and contractures, and also the atrophy of the muscles, are less pronounced; the electric irritability disappears slowly. The reflexes are usually diminished. There are more or less violent pains in the back, trunk and extremities, dysæsthesiæ and paræsthesiæ and later on marked anæsthesia, paralysis of the sphincters, and bed-sores. The clinical picture is consequently essentially that of Duchenne's "paralysie générale spinale diffuse subaiguë," the individual cases, of course, presenting countless modifications, according to the stage and the extent of the process.

The clinical history of *myelitis chronica disseminata*, or multiple sclerosis, which will be described in detail in another place (No. 11), is in some cases very characteristic, and in others just the contrary. The reason of this is that the grouping and character of the symptoms depend entirely on the accidental locations of the different centres of disease. The numerous, concomitant cerebral symptoms, the psychical disturbances, the peculiar impairments of speech, and the affections of different cerebral nerves, are particularly important; so are also the very irregular distribution of the paralyses, the peculiar and varying range of the anæsthesia, the trembling of the limbs during voluntary movement, etc.

That form of chronic myelitis which is confined to the posterior white columns, and is usually designated as *tabes dorsalis* (gray degeneration or sclerosis of the posterior columns), will be described in detail in a subsequent chapter (No. 12). It is characterized, in the early stages, by severe lancinating pains, slight disturbances of sensation, a girdle-sensation, and not unfrequently by implication of the optici and the nerves supplying the ocular muscles; in the later stages, by impairment of co-ordination (ataxia), weakness of the bladder and of the sexual functions, loss of the tendon-reflexes, etc.

An essentially different clinical history is, on the other hand, presented by that form of chronic myelitis called *lateral sclerosis*, in which the morbid process can with great probability be located in the lateral columns. Here we have a motor weakness, which gradually increases in intensity and range, and is accompanied by striking muscular contractions and contractures, and by great increase of the tendon-reflexes. There is no ataxia, but instead of it a markedly spastic gait; no disorder of sensation, no atrophy of the muscles, no diminution of their electric irritability, no paralysis of the bladder, no decubitus. This form, too, must be made the subject of special consideration (see No. 13).

Finally, it is very difficult to recognize during life that form of myelitis which is localized in the peripheral layers of the white substance, and which is perhaps most correctly designated as *myelomeningitis chronica* (the sclérose corticale of the French). It may be recognized in a few cases by the fact that the symptoms of chronic meningitis (see p. 255) are accompanied by somewhat more pronounced manifestations of both sensory and motor paralysis. It is said that muscular atrophy, as a rule, does not occur. These points are not very safe or reliable from a diagnostic point of view.

Diagnosis.

The diagnosis of the different forms of chronic myelitis is to a great extent evident from the preceding descriptions, and for further details we may refer to the special chapters which follow. We must make place here, however, for a few words concerning the simple, chronic myelitis transversa. It is characterized by slow development of paraplegia with relatively slight irritative symptoms, but with more or less marked disturbances of sensation and of the bladder; usually also with muscular contractions, increased reflexes, and decubitus. Hence, all, or at least most of the paths in the cross-section of the cord are included in the lesion, though with varying degrees of intensity. The process, however, is limited to a relatively small area in a vertical direction, a fact which can be most readily recognized from the state of the reflex irritability. The affection is but slightly progressive in character, and runs a tedious course. The diagnosis of transverse myelitis can readily be made from these points, all the more certainly if the existence of such etiological influences as slow compression, syphilis, and the like, can be demonstrated. The height at which the lesion is located in the cord can usually be easily determined from the range of the paralysis, the state of the reflex and electric irritability and of the nutrition, etc.

The differentiation of transverse myelitis from the other forms of the affection, from the later stages of tabes, from lateral sclerosis, from multiple sclerosis, and also from progressive muscular atrophy and from paralyses of the cauda equina, is in most cases easy. Still there are many cases of peripheral paralysis due to affections of the plexus or of the cauda equina, to compression of a number of the nerve-roots, etc., in which the differentiation from chronic myelitis is very difficult. In the diagnosis of such cases, the co-equal extension of the motor and sensory disturbances, the absence of all reflexes, the loss of electric irritability, the limitation of the disturbances to certain isolated nerve-territories, etc., may be turned to good account.

There still remain, however, numerous complicated and perhaps irregular cases, in which an exact diagnosis of the special form of the disease is downright impossible. This leads us to say a few words concerning a point to which, despite its great importance, too little attention has been paid in practice, and to the disregard of which a great part of the obscurity and confusion in the accepted clinical conceptions of the disease is due. It is the simple fact, that the myelitic processes, described in the preceding pages as strictly localized in particular parts of the cross-section of the cord, do not by any means always remain limited to these particular parts, but often extend beyond them and involve neighboring parts; that, in other words, in addition to the special forms of chronic myelitis, transition-forms exist and combinations occur which are of course characterized by combinations of the clinical histories. While, in the very great majority of the cases, sclerosis of the posterior columns gives rise to the typical clinical picture of tabes, while the regular form of sclerosis of the lateral columns, as a rule, remains limited to the lateral columns and produces its own special symptoms, while poliomyelitis anterior chronica has its own distinct clinical history, we meet in practice with not a few cases in which the typical symptoms of tabes are combined with those of sclerosis of the lateral columns, as well as cases in which the symptoms of a poliomyelitis anterior are added to those of a lateral sclerosis or of a tabes. Pathological anatomy has already furnished a sufficient explanation of this fact. A number of reliable observations have been recorded, in which the anatomical examination demonstrated the co-existence of gray degeneration of the posterior and of the lateral columns, or the combination of an extensive lesion of the anterior gray substance with sclerosis of the posterior or lateral columns. Careless investigations and an insufficient acquaintance with these facts have naturally led to the incorrect conclusion, that lesions of special parts of the cross-section of the cord do not give rise to special groups of symptoms, but that on the contrary these lesions can cause sometimes one and sometimes another set of symptoms. The knowledge which is now possessed of the pathology of the cord, teaches us, however, that each typical clinical picture must correspond to some distinct anatomical lesion, and that where we meet with a combination of different clinical pictures, we must also assume a combination of different anatomical lesions. Valuable confirmation of this statement is furnished by the striking conformity of the typical cases of each particular form of the disease. These combined forms are not at all rare, although they certainly constitute but a small minority in comparison with the pure, typical cases of each form. It is true, that we cannot hope to be able to recognize these typical clinical pictures whenever met with, even when more or less confused by combination with one another, without a careful observation and study of an abundant material. Without this preparation, the differential diagnosis of such cases will prove very troublesome, and we shall have reason to complain of the obscurity and ambiguity of the clinical histories, while with it we shall be able to take a more decided stand, and to resolve the combination pictures more readily into their various component parts. That every case of this sort requires a special diagnosis, and that countless modifications are possible, are self-evident facts. We will return to these points again when speaking of the individual forms of the disease.

Prognosis.

The prognosis varies very greatly in the different forms of chronic myelitis; for details, we may refer to the appropriate chapters. In simple chronic myelitis transversa the prognosis is in general unfavorable. The affection is always a severe and a dangerous one, and the best that can be hoped for is recovery with a heritage of considerable residua. A real and complete recovery can only be looked for in exceptional cases, where the disease is very mild and has developed in otherwise healthy persons, and where, moreover, treatment has been begun in the earliest stages.

Only rarely, also, will it be possible to predict a partial recovery, followed by a persistence of the status quo for many years or until the natural end of life. In such cases a complete recovery of power over the limbs can never be expected. In the majority of the cases a slow, progressive course must be looked for, and usually only a few years of life can be promised to the patients.

The prognosis will be partly determined or influenced by the more or less progressive and ascending character of the disease, by the tendency to aggravations and relapses which sometimes exists, by the effect produced by certain therapeutic measures, and finally, by the more secondary symptoms, particularly the cystitis and decubitus which most frequently place life in danger. None of these points require a more extended consideration.

Treatment.

Is there any *prophylaxis* for chronic myelitis? Unquestionably, though only to a certain, very limited degree. The precautionary measures, which in general prove useful in preventing the development of diseases of the cord, are serviceable here also. Their employment is advisable particularly in the case of the numerous individuals, who belong to neuropathic families, and more especially when affections of the spinal cord have occurred in the generations immediately preceding. In such individuals, more even than in others, the most beneficial effects will be derived from a rational *hardening of the body* (by means of cold sponging, baths, gymnastics, exercise in the open air, pedestrian excursions, nourishing diet, proper but not too warm clothing, etc.), and from appropriate *hygiene of the nervous system* (proper alternation of rest and work, avoidance of mental overexertion and of violent emotional excitement, sufficient sleep, etc.). The avoidance of sexual excesses, of spirituous drinks and other irritating articles, of bodily overexertion, of the causes of catching cold, etc., will also assist to a certain extent in keeping the spinal cord in a state of health. Unquestionably, much would be gained in many of these cases, if the physician could induce the individuals in question to follow out strictly the above precautionary measures. Unfortunately, however, we can rarely succeed in this.

In respect to the *causal indication* in chronic myelitis, it is only in isolated cases that anything can be done. In this connection, the importance of a careful treatment of acute myelitis, continued until recovery is complete, must be alluded to. In other cases there may be some compressing agent (caries of the vertebræ, new growths, etc.), which we must endeavor to remove; or a syphilis, which forms the basis of the spinal affection, must be energetically attacked; or diseases of peripheral organs, of the intestines, or of the urinary or sexual organs, must be subjected to proper treatment. When, in addition to hemorrhoids, a tendency to constipation, dyspepsia, etc., exist, the appropriate treatment for them should be employed; the favorable results obtained in many cases of chronic myelitis (so-called hemorrhoidal tabes), by a course of treatment at saline springs, may perhaps be accounted for by its influence on these derangements. Usually, however, in consequence of the slow and lingering manner in which the disease is developed, it is impossible to discover any causal indication, or, if discovered, to carry it out.

For the treatment of the disease when it has developed to a certain degree, we possess, it is true, numerous remedies, but we must not deceive ourselves with regard to their efficiency. Chronic myelitis, in all its different forms, is a disease from which recovery is rare, and in the best cases slow. The earlier the treatment is begun, the better are the prospects of a favorable termination. Hence, it is particularly important, that notice should be taken of the earliest and even the most trifling of the initial symptoms, and an energetic and appropriate treatment at once commenced. It is better to be too careful and anxious, and to ascribe excessive importance to trifling and perhaps harmless symptoms, than to suffer the patient and ourselves to be deceived concerning the gravity of the initial symptoms, and thus give the creeping evil time to develop into a dangerous and unconquerable foe.

We shall rarely have an opportunity to employ *antiphlogistic measures*. The development of the disease is much too lingering to permit their employment; the symptoms of hyperæmia and irritation remain more in the background. Hence, blood-letting, ice, confinement to bed, drastic cathartics, depressing diet, and the like, should be avoided. These measures will only be indicated, when subacute or acute exacerbations occur. Under such circumstances, the treatment laid down for acute myelitis should be employed.

The much used and often misused *derivative measures* will naturally be the next to be thought of. Unfortunately, experience teaches that they prove, as a rule, of very little use. In former times they were invariably employed, and in a very energetic manner; the red-hot iron was an especial favorite, and all sufferers from spinal disease bore the marks of it in numerous cicatrices on the back. The results produced by these measures were entirely disproportionate to the frequency and energy with which they were employed. The persistent employment of milder derivatives (repeated flying or suppurating blisters, pustulating salves, painting with iodine, etc.) has also proved, as a rule, of little use. Personally I do not remember to have ever obtained any distinct advantage from the employment of these remedies, though, it is true, my experience with them has not been particularly extensive. Our attention is, however, directed to them over and over again by the favorable results they produce in other diseases, and perhaps fresh trials of them are now more admissible, in view of the facts that we can diagnose the different forms of the disease more positively, and can also pick out more readily the cases which present any chance of success. Such trials, however, should be carried out in a systematic manner, and with exclusion of other remedies.

We recommend as a relatively harmless, and still in many cases decidedly efficient derivative measure, the application of dry cups along the spine, repeated every few days. Brown-Séquard praises highly the daily employment of a hot (37–40° C., 98.5°–104° F.) douche to the back, for two or three minutes at a time; the stream should be nearly an inch in diameter.

In the treatment of chronic myelitis at the present day (we are speaking now of all the forms together, referring to the special accounts of the different forms for further details), the chief reliance is placed on three curative agencies. These are *baths* (particularly the thermæ, hot and cold brine baths, and to a less degree steel-baths), *hydropathy*, and *galvanism*. We have already (in the General Part, p. 164 et seq.) spoken at length of these things, and endeavored to determine with some degree of accuracy the general indications for their employment, and we have only to add here a few points bearing directly on their use in chronic myelitis.

In order to attain an approximately correct idea of the action of these agents, I have made out a table from the cases of chronic myelitis (in all its forms, inclusive of multiple sclerosis, lateral sclerosis and sclerosis of the posterior columns), that have come under my own observation, of which I have notes of the treatment and its effects. This table includes 175 different observations that were made on 107 different cases. It is true that this material is not homogeneous. It contains a large number of hospital cases, and many old and severe cases that defied all treatment; most of them were treated solely by electricity; many of them have undergone several courses of treatment, and have thus afforded opportunity for repeated observation. With all this, however, some important points, which I would gladly see made the subject of further investigations, can be gathered from this table.

In the first place, the result of my collation of facts is decidedly unfavorable for the *indifferent thermæ* (Wildbad, Teplitz, etc.). Out of twenty-two cases in which these thermæ were employed, there were no less than twelve which were decidedly injured by them; in these cases an aggravation of the disease set in during or immediately after the course of treatment by baths. Of the remaining ten cases, seven experienced no change at all, and only in three was there any improvement; one of these was cured. In two of the three cases which were improved, the baths were, however, cooler, of shorter duration, and less numerous. These observations coincide with the experience of most specialists. The results are at all events not encouraging; they take away all desire to send such patients to the thermal springs, which was formerly the universal custom, and is even yet frequently done. If the physicians located at watering-places could be induced to publish unprejudiced and careful observations, based on correct diagnoses, a great desideratum would be fulfilled. At all events, I no longer risk a trial of the thermæ in these cases, except when I have guarantees for the most cautious employment of a modified course of baths. The temperature of the bath should never be above 33°–34° C. (91⅜–93⅛° F.), and prolonged immersion should be avoided. If I were to attempt to pick out the actual cases for which the thermæ are suitable, I should find myself in a difficult position. It is usually stated that the cases with prominent irritative symptoms, with hyperæsthesia, with meningitic symptoms, etc., are especially benefited by them. I must confess, however, that without further critical observations, I cannot regard these indications as reliable. At present I would only recommend a trial of hot baths after other remedies have failed, and even then only with every precaution.

My experience has been much more favorable with the *thermal brine baths* (Rehme, Nauheim). I possess notes of twenty-one cases; of these twelve were improved by the course of treatment, nine received no benefit from it, but none were rendered worse. Here, too, I am convinced that much depends on the manner in which the treatment is carried out, on the temperature and duration of the baths. It is advisable that they should be of a moderate temperature (30–26° C.—86–78⅘° F.) and not too prolonged, and that the water should not contain an excessive quantity of carbonic acid. Any increase in the activity of the treatment must be made with great caution. These baths are suitable for most of the cases. At the most, I would hesitate to recommend them for cases attended by great irritability, violent pains and other symptoms of irritation.

I have had no experience at all with the ordinary *brine-baths*, and very little with *chalybeate* and *turf baths*. The latter proved useful in two cases, and detrimental in one. They are highly extolled by many authors. In Franzensbad particularly, various good results have been obtained by their use. This, too, is a subject worthy of further investigation.

Concerning *animal-baths*, *hot sand baths*, *pine-needle baths*, *steam-baths*, *hot-air baths*, etc., I know very little from personal experience. In most of the cases they are superfluous, and, when used at very high temperatures, they are actually injurious and dangerous.

On the other hand, the results of a rationally conducted *cold-water cure* are exceedingly favorable. Out of twenty-nine cases of which I have notes, the results of the treatment were favorable in twenty-one, negative in five, and unfavorable in three. Here too, of course, everything depends on the manner in which the cold water is used. I cannot insist too much on the danger of forced cures, of all severe and strongly exciting procedures, such as the employment of water at very low temperatures, douches, sharp slappings, etc. These measures are, as a rule, absolutely injurious in cases of myelitis; even wet packs of the entire body have, to my surprise, generally proved injurious. Simple rubbing with wet cloths, foot-baths and sponging the back, hip-baths, half-baths with affusions to the back, local compresses to the back left on till they become warm, etc., seem to be the measures which are chiefly applicable. The treatment should always be begun with moderate temperatures (20–25° C.—68–77° F.), and we should never go below 16–12° C. (60⅞–53⅝° F.). I believe also that excessive prolongation of the treatment is injurious. Many over-zealous hydropathists are only too often led, by their great confidence in the water-cure, to prolong the treatment to an extent that proves detrimental to the patients.

The water-cure is, all things considered, one of the most important and most promising means of treating chronic myelitis. The mistrust with which it is regarded by some authors is, as far as my own experience goes, entirely unjustifiable. It is suitable for almost all cases, though of course the method of application must vary according to the peculiarities of the individual cases. Some patients, however, cannot bear the treatment at all; it is followed by insufficient reaction, causes chilliness, discomfort, etc. In such cases the treatment should not be persisted in. The choice of the establishment is of course a matter of great importance. I prefer one situated in a healthy mountainous locality and in a wooded region. The chief requisite, however, is always the presence of an intelligent and competent physician.

In recent times the *galvanic current* has acquired a prominent position in the therapy of chronic myelitis. Out of one hundred cases treated by it, I obtained a more or less favorable result in fifty-two. Complete recovery ensued, it is true, only in a few of these cases, but a very considerable and striking improvement was observed in quite a number of them; in most of them, however, the improvement was only moderate. In forty-seven of the cases the results were negative, although in some of them the treatment was persisted in for a very long time. In only one case was the effect of galvanization decidedly bad.

The chief part of this treatment is, of course, the direct galvanization of the cord, which must be conducted precisely according to the principles which have been exhaustively considered in another chapter (see page 178). The electrodes must be applied differently, in accordance with the differences in the position and extent of the focus of disease. It is best to let both poles act successively, either with a stabile or with a slowly labile current. When we have to deal with band-shaped sclerosis, it is advisable to include the cervical sympathetic also within the sphere of the electricity. The current used should not be very strong, and the séances should be short. I cannot insist sufficiently on the point, that the treatment, to be successful, must be conducted with great care and skill. Great patience on both sides is, as a rule, necessary. The treatment must be continued for months, being occasionally interrupted only to be recommenced after a brief pause. It is expedient to alternate or combine galvanization with other methods of treatment.

It is as yet impossible to indicate the cases for which the galvanic treatment is especially suitable. It may, indeed, be tried in most or all cases, as we know of no positive counter-indication. Every now and then, however, we shall meet with patients who cannot bear, or who think they cannot bear, even the most cautious application of the galvanic current. In such cases the treatment must be at once discontinued.

Little can be expected from *internal medicines* in the treatment of chronic myelitis. *Nitrate of silver*, which was first recommended by Wunderlich, is the most reliable of all of them. In a few cases it has undeniably proved beneficial. It is impossible to give the exact indications for its employment, but, as it is in general a harmless medicine, it may be tried in most of the cases. Brown-Séquard very urgently recommends *ergot* and *belladonna*, both of which are said to lessen the congestion of the spinal cord and to diminish its reflex irritability. He gives the ergot in doses of 0.30–0.50 (gr. v.–viiss.) twice a day, and in connection with it applies a large belladonna plaster to the back, or gives extract of belladonna internally in doses of 0.015 to 0.02 (gr. ⅕–⅓) twice a day. The effect is said to be very decided in many paraplegias. *Iodide of potassium* has rather unexpectedly proved almost invariably useless. I at least, often as I have prescribed it, have never seen any decided improvement follow its use. Even in the paraplegias of syphilitic patients, its curative action is often very limited. *Arsenic*, too, has in my experience, as a rule, proved useless. *Phosphorus* I have seldom prescribed; its curative action seems to me dubious, and its administration not entirely without danger. *Strychnine* is in my opinion almost always decidedly objectionable in chronic myelitis. In the earlier stages it is more liable to do harm than good, and for the later stages, when the residua of the myelitis (paralyses, etc.) are to be treated, we possess in electricity a much more reliable as well as safer remedy.

In consequence of the long duration of the disease, the physician will often find himself tempted to employ in a tentative manner, not only the above, but also numerous other remedies. It will be fortunate both for physician and patient, when the right remedy is discovered at once.

The *general management*, *the diet* and the *mode of life* of the patients are in our opinion of very essential importance. These points are still very often sinned against, partly through ignorance, partly through carelessness. Above all, rest and a regular mode of life are essential; overexertion of every kind, mental as well as bodily, must be avoided; sexual intercourse should be confined within the strictest possible limits or completely stopped; all excitement, violent emotions, etc., must be prevented as much as possible. The *diet* must be simple and easy of digestion, but at the same time nutritious. Many patients are, however, benefited by a very abundant supply of nourishment, and by cod-liver oil. Spirituous drinks should only be allowed in small quantities. Strong coffee and tea and strong cigars must be avoided. It is very important that the bowels should be kept regular; for this purpose we must usually employ both aperients and clysters. The patients should be strongly urged to keep as much as possible in the open air. When practicable, a prolonged sojourn in a mountainous region at a moderate elevation, or at the sea-side, will be useful; it is also advisable that the winters should be passed in the South, in the Riviera, the southern Alpine valleys, etc. The regulation of the bodily exercise is usually a very important question. Much harm can easily be done by exceeding the permitted amount of exercise. I have repeatedly known patients, who had begun to improve, to be thrown back for a long time by an unduly prolonged walk. The idea which is entertained by many patients, that a commencing muscular weakness and stiffness can be removed by forced exertion of the muscles, must be energetically combated. I believe that patients who are still capable of moving about, should be allowed to take only so much exercise as they can indulge in *without fatigue;* as soon as they *begin* to grow tired, they must rest for a time, and may then resume the walk or other movement.

In many cases, it is true, exercise is out of the question—the patients are bed-ridden, paralyzed. Such patients must make it a point not to lie always and exclusively on the back. Brown-Séquard wants to discard the dorsal position entirely, and to replace it by the lateral or abdominal position, but it is more than doubtful whether it will be possible to do so. For these paraplegic patients a wheel-chair may be used, though it is but an unsatisfactory substitute for walking exercise. It will at least enable them to enjoy the fresh air regularly and freely.

In most of the cases of chronic myelitis, there are also a number of *symptomatic indications* that require attention. The most important of these is unquestionably the *prevention of cystitis and decubitus;* the closest attention must be paid to this point under all circumstances. We have already discussed it at length on page 193 et seq., to which the reader is referred.

Another frequent and important indication is the *relief of the pains*—of the lancinating pains of tabes, the spontaneous and radiating pains of myelitis transversalis, etc. These will very frequently resist all imaginable remedies, and prove a source of great annoyance to the physician, as well as of torment to the patient. The most rapid relief will always be obtained from hypodermic injections of morphine, but the dangerous consequences of the constant use of this drug must deter us from too frequent employment of the injections. After morphine the most serviceable drugs are bromide of potassium, quinine, bromide of quinine, zinc, and valerian. Cutaneous irritation, electricity (the faradic brush or galvanic current), Preissnitz's compresses, applications of chloroform, and frictions with veratrine, oil of hyoscyamus, and the like, often do good service. Similar remedies are employed with more or less success to relieve the manifestations of motor irritation, the painful spasms, reflex twitching, etc. For the paralyses, atrophies, anæsthesiæ, etc., which persist after the disease has run its course, electricity is the sovereign remedy.

The *general plan of treatment* must of course, apart from the stage of the disease, be regulated chiefly according to the individuality of the case, or the internal and external conditions of the patient. Any causative influences that may be discovered must be first of all removed, and the surroundings of the patient and his mode of life regulated. In the relatively recent cases, which are still in the early stages, the following course of treatment may be recommended after the above points have been attended to: mild, but nevertheless efficient hydropathic treatment, in conjunction with the employment of the galvanic current; in appropriate cases these may be combined with mild derivatives (blisters, dry cups, Preissnitz's compresses, etc.), change of air, and a sojourn in the mountains or at the sea-side. As the disease progresses a course of treatment at the hot brine-springs, or in a hydropathic establishment may be tried, alternating with the treatment by galvanism; internally the persistent use of nitrate of silver.

In the older cases, where the prospects of recovery are much less, a cautious alternation of the different methods of treatment is requisite. During the winter such patients should be treated by galvanism, and should also be made to employ cautiously cold applications combined with frictions. In the summer the thermal brines, cold-water treatment in the mountains, and perhaps also the thermæ, may be tried. In addition to these measures, various internal remedies may be tried, the symptomatic indications must be carefully fulfilled, and decubitus and cystitis must be guarded against.

Under all circumstances, the employment of psychical influences constitutes an important part of the treatment of chronic myelitis. The courage of the patient must be maintained, his confidence in the various methods of treatment strengthened, and his hopes constantly aroused and reawakened. Unfortunately the physician must in only too many cases rest contented when he can succeed in this task, which is itself by no means a light one, and brighten the miserable existence of the patients by an occasional glimmer of fresh hope.

10. *Simple Softening of the Spinal Cord.—Myelomalacia.*

Brown-Séquard, Lectures, etc., on Paralysis of the Lower Extremities. p. 34. 1861. —*Hennig*, Fall von Erweichung des Rückenmarks. Arch. d. Heilk. I. S. 188. 1860.—*Leyden*, L. c. II. S. 38, 51 ff.—*Panum*, Experim. Beitr. z. Lehre von d. Embolie. Virch. Arch. Bd. 25. S. 308. 1862.—*Hasse*, L. c. 2 Aufl. S. 706.—*Hammond*, L. c. 3. edit. 1873. p. 463.—*Willigk*, Ueber d. Verhalten d. Nervenzellen bei embol. Processen im R.-M. Prager Vierteljahrschr. 1875. III. S. 41. —*D. J. Hamilton*, On Reflex Paralysis and Urinary Paraplegia. Brit. and For. Medico-chir. Rev. Tome I. VII. p. 440. 1876.—*Em. Bertin*, Ramolliss. d. l. moëlle. Dict. Encyclop. des. Sc. méd. II. Sér. T. VIII. p. 751. 1854.

It may perhaps seem venturesome in the present state of our knowledge, to devote a special chapter to simple, non-inflammatory softening of the spinal cord. We are still deplorably ignorant with regard to this affection, and hence the chapter will necessarily be a short one. That there really is, however, a non-inflammatory as well as an inflammatory softening of the spinal cord, must unquestionably be admitted; it is demonstrated both by convincing analogies with similar processes in the brain, and by a certain number of anatomical and clinical facts. It is in order now to describe as accurately as possible, the anatomical changes and clinical history of this simple softening, in order to differentiate it more positively from the inflammatory affection.

It seems to us particularly desirable to protest against the misuse of the word "softening," which is at present very commonly applied to simple and even to acute inflammation. The terms softening and inflammation are by no means synonymous; not every softening is the result of inflammation, and not every inflammation leads to softening. The latter certainly occurs as a concomitant and a consecutive lesion in many forms of spinal inflammation, but it is by no means an essential part of them. We might, with just as much and perhaps even more reason, employ the term "hyperæmia," or "exudation," or "fatty degeneration," to designate inflammation.

It is unquestionably better to discard entirely the use of the term "softening" for inflammatory conditions; this would be the best way to avoid misunderstandings. If used at all, it should be reserved for the simple, non-inflammatory processes of softening, which are undoubtedly met with in the cord; for these, it seems to us perfectly appropriate.

It is true that the existence of these non-inflammatory changes has not yet been demonstrated with that degree of positiveness, which is desirable, and that we do not, as in the case of the brain, possess experimental proofs or clinical observations equivalent to them (cerebral emboli) for the most important forms of the softening. With respect, moreover, to the histological processes, and more particularly the points which distinguish the simple from inflammatory softening, we are as yet to a great extent in outer darkness. Still, it seems to us that there has been too great a tendency in recent times to class under myelitis all lesions of the spinal cord characterized by softening, even though the characteristic histological evidences of inflammation be by no means always present.

A few brief remarks concerning simple softening may be made now, principally with a view to incite others to further studies and investigations.

The *pathogenesis* of the affection is enveloped in numerous obscurities. The softening which is due to *thrombosis* and *embolism* is probably the easiest to explain, in consequence of its analogy with the corresponding cerebral process. The effects of embolism of the abdominal aorta, from which we might expect the most valuable information in this connection, have not yet been sufficiently studied. It has been stated, that white softening of the cord has been found as one of these effects.

Embolism of the small arteries of the cord is very rare, and would at all events produce only very circumscribed centres of softening. Panum, Vulpian and others have studied it experimentally, and have demonstrated the development of small centres of red softening as the result of such emboli. Leyden found small, inflammatory foci produced by emboli, which were derived from ulcerative endocarditis. Willigk, in his recent investigations, discovered no centres of true softening in the neighborhood of the obstructed vessels, but found instead of softening, in some cases a hyperplasia of the connective tissue with atrophy and destruction of the ganglion-cells, and in others a peculiar "colloid" degeneration of the ganglion-cells without increase of the connective tissue. All this requires further study and investigation. The case of multiple thrombosis of the small vessels of the cord, reported by Hamilton, is a remarkable one.

There is little reason to doubt that a severe *affection of the walls of the vessels*, such as atheroma, calcification, or sclerosis, diminution of the vascular lumen, etc., can also cause similar retrogressive metamorphoses in the tissue of the cord, which finally terminate in softening.

Second in order of importance comes the *softening produced by slow compression of the cord.* We have already (p. 323) stated, that a true myelitis (compression-myelitis, inflammatory softening) of the affected portion of the cord is almost without exception excited by such compression. Nevertheless, exceptions do apparently occur. The fact that in not a few cases of compression of the cord, the microscope reveals in the softened mass no granule-cells, no hyperplasia of the connective tissue, no proliferation of the nuclei, but only swollen and disintegrated nerve-elements, speaks unmistakably in favor of the view that the process is sometimes, at least for a time, one of simple softening. There is nothing forced in the theory, that the ischæmia of the cord caused by the compression results in simple softening, and that this condition may persist as long as the compressing mass does not develop strongly irritating qualities, and implicates the cord only very gradually and slowly. This, too, requires verification.

It is still very doubtful whether a *spontaneous simple softening* ever occurs. At all events, the older observations which are adduced in favor of it, are by no means conclusive, since they lack the microscopical examination which alone can determine positively the absence of inflammatory changes.

A *senile softening* of the cord is spoken of, but we must confess that the supposed examples of it contained in medical literature do not by any means convince us of its actual existence. Still, it is certainly possible that the vascular affections, which are so common in old age, may occasionally give rise to small spots of softening in the spinal cord. At all events, however, the fact requires more accurate authentication.

We may mention, finally, that Hammond assumes *sexual excesses* to be the cause of simple softening in a few cases.

Under these circumstances there is but little that can be said concerning the special *pathological anatomy* of myelomalacia; there is always danger of confounding the inflammatory with the non-inflammatory foci of softening. In fact, macroscopically they resemble each other very closely. The spinal cord is soft and pulpy, and swells up above the level of the cut; in pure cases it is white, and in the later stages becomes creamy, milky, and fluid. Pronounced red softening can, however, also occur in consequence of the blood being dammed back in the ischæmic vascular territories. When the fatty degeneration assumes special prominence, the softened spot may acquire a more yellowish color, and present the appearances of yellow softening. At its boundaries, the softened spot usually merges imperceptibly into the normal tissue.

It is difficult to decide whether the case of Hennig should really be classed as simple softening; if it must be, it would present a sort of gelatinous or colloid degeneration, with fatty degeneration of the vessels.

The microscopical examination can alone furnish conclusive evidence of the inflammatory or non-inflammatory nature of the softened spot. Unfortunately, however, we do not as yet possess a sufficient number of accurate reports bearing on this point. Wherever we find a large number of cells containing fat granules, tensely distended blood-vessels, numerous young cells, increase of the interstitial tissue, swollen axis-cylinders, etc., we are justified in assuming positively the inflammatory character of the process. On the other hand, when these characteristics are wanting, and we only find simply swollen and disintegrated nerve-fibres, ganglion-cells in a state of glassy swelling, a few cellular elements and fat-granule-cells, and a small quantity of fatty detritus, we must diagnose a simple softening. Further and searching investigations of this point are, however, very much to be desired.

The *symptomatology* of simple softening is also exceedingly obscure. In the clinical histories we meet with the picture of a more or less acute or subacute myelitis unattended by fever, and presenting only a few non-essential modifications, that are of little help in the diagnosis. According to Hammond, the absence of motor symptoms of irritation is particularly distinctive of simple softening. That this, however, does not hold true of all cases without exception, is demonstrated by the histories of many cases of acute myelitis.

In the commencement of the disease a sensation of numbness and a feeling of weakness are experienced, which gradually become more intense, but are not accompanied by pains, muscular twitchings, etc. Progressive weakness of the bladder is added to the paralysis; the anal sphincter is also affected, and the reflex excitability seems diminished.

In the later stages of the affection we have the complete picture, modified only in accordance with the localization of the softened spot, of a severe sensory and motor paraplegia, with paralysis of the sphincters, bed-sores, and the well-known termination.

According to Brown-Séquard and Hammond, the absence of motor and sensory signs of irritation is conclusive for the *diagnosis* from inflammatory softening. According to them, there are no pains, no spasms, no hyperæsthesia, and no increase of the reflex activity. Personally, however, we cannot suppress a slight doubt as to the pathognomonic importance of the absence of these symptoms, and feel ourselves forced to look to the future for the possibility of a more exact diagnosis of simple myelomalacia.

The *prognosis* of the affection must be decidedly unfavorable, when the centres of softening are at all extensive. The very small foci, which give rise only to a very limited array of symptoms, or perhaps to none at all, do not, as a rule, call for a prognosis.

The *treatment* of simple softening can also be disposed of in a few words. It must be carried on essentially according to the rules laid down for the non-febrile forms of myelitis without prominent inflammatory symptoms.

Brown-Séquard recommends, besides iron and quinine, chiefly iodide of potassium, which is best administered in a bitter decoction (cinchona, colombo, rhubarb); strychnine only with the greatest caution; *no* ergot! no belladonna! He believes, further, that the cold douche or shower-bath to the back is indicated, as is also confinement to the dorsal position.

In general, abundant and nourishing food, when possible moderate exercise, and the employment of the galvanic current, will be found advantageous.

11. *Multiple Sclerosis of the Spinal Cord (and Brain).—Multiple, nodular Sclerosis; disseminated, multilocular, insulated Sclerosis of the Central Nervous System.—Sclérose en plaques disséminées.—Insular Sclerosis.*

Cruveilhier, Anat. pathol. Livr. 32, 38. 1835-42.—*Charcot*, Klin. Vortr. über die Krankh. des Nervensyst. Deutsch von *Fetzer*. 1874.—*Hasse*, l. c. 2. Aufl. S. 708.—*Rosenthal*, l. c. 2. Aufl. S. 147.—*Hammond*, l. c. 3. Edit. p. 637.—*Leyden*, l. c. II. S. 375.—*Bernheim*, Dict. encyclop. des Sc. méd. II. Sér. T. VIII. p. 707.

Frerichs, Ueber Hirnsklerose. Haeser's Arch. X. S. 334. 1849.—*Valentiner*, Ueber Sklerose des Gehirns u. R.-M. Deutsche Klin. 1856. No. 14 bis 16.—*Leyden*, Ueber graue Degen. des R.-M. ibid. 1863. No. 13.—*Rindfleisch*, Histol. Detail zur grauen Degen. Virch. Arch. Bd. 26. S. 474. 1863.—*W. Zenker*, Sklerose des Gehirns u. R.-M. Zeitschr. f. ration. Medic. 3. Reihe 24. Band. 1865.—*Vulpian*, Notes sur la sclérose en plaques, etc. L'Union méd. 1866. Nr. 67-72.—*Charcot*, Gaz. des hôp. 1868. Nr. 102, 103.—*Ordenstein*, Sur la paralys. agit. et la sclér. en plaques généralisée. Thèse. Paris, 1867.—*Bourneville*, Mouvem. méd. 1868. Nr. 13-25, u. 1869, Nr. 27 bis 33.—*Bourneville et Guérard*, De la sclérose en plaques dissém. Paris, 1869.—*Guérard*, Essai sur la scl. en plaques. Thèse. Paris, 1869.—*Liouville*, Gaz. méd. 1870. Nr. 19 u. 20.—*Joffroy*, ibid. Nr. 23, 24.—*Magnan*, ibid. Nr. 14. p. 183.—*Leo*, Beitr. zur Erk. der Sklerose des Gehirns u. R.-M. Deutsch. Arch. für klin. Medic. IV. 1868.—*H. Schuele*, ibid. VII. S. 159. 1870, u. VIII. S. 223. 1871. *W. Leube*, ibid. VIII. S. 1. 1870.—u. Klin. Ber. aus dem Krankenhaus zu Jena. 1875. S. 111.—*Zenker*, Deutsch. Arch. f. klin. Medic. VIII. S. 126. 1870. —*Ebstein*, Spinale Form der multipl. Sklerose. ibid. IX. S. 528, u. X. 595. 1872.—*Kelp*, Ueber Hirnsklerose. ibid. X. S. 224. 1872.—*Buchwald*, ibid. X. S. 478. 1872.—*Otto*, ibid. X. S. 531. 1872.—*Berlin*, ibid. XIV. S. 103. 1874. —*Engesser*, ibid. XVII. S. 556. 1876.—*Baerwinkel*, Arch. d. Heilk. X. S. 590. 1869.—*C. Hirsch*, Deutsch. Klin. 1870. Nr. 33-38.—*Radlick*, Fall von Sclérose en plaques. Diss. Berlin, 1874.—*Pohl*, Sklerose des Gehirns u. R.-M. Diss. Berlin, 1874.—*Westphal*, Ueber eine Affection des Nervensyst. nach Pocken u. Typhus. Arch. f. Psych. u. Nerv. III. S. 376. 1872.—*The same*, Neue Charité-Annalen. I. Jahrg. 1874. S. 427.—*Otto*, Casuist. Beitr. zu den nervös. Nach-

krankh. der Pocken. Virchow-Hirsch, Jahresbericht pro 1872. II. S. 28.—*Jolly*, Ueber multiple Hirnsklerose. Arch. f. Psych. u. Nerv. III. S. 711. 1872.—*Chvostek*, Wien. med. Pr. 1873 u. 1874.—Allgem. Wien. med. Zeit. 1875. Nr. 11-20.—*W. Moxon*, Eight Cases of Insular Sclerosis. Guy's Hosp. Rep. XX. p. 437. 1875.—*Mollière*, Sur quelques points du diagnost. d. l. sclér. des centr. nerv. Lyon méd. 1875. Nr. 28.—*F. Schultze*, Ueber das Verhältniss d. Paralys. agit. zur multiplen Sklerose des R.-M. Virch. Arch. Bd. 68. 1876.—*Christidis*, Ueber multiple Sklerose, etc. Diss. Würzburg, 1875.—*E. Killian*, Ueber einen Fall von diffuser Myelitis chronica. Arch. f. Psych. u. Nervenkrankh. VII. S. 28. 1876.—*Putzar*, Fall von multipl. Sklerose des Gehirns u. R.-M. Deutsch. Arch. für klin. Med. XIX. S. 217. 1877.

History.

The disease now generally known under this name has only figured in the annals of science for a few decades, and only became thoroughly understood during the last decennium. The first minute account of the affection appeared, accompanied by excellent illustrations, in the large atlas of Cruveilhier (1835 to 1842). A similar case, observed by Tuerck in the year 1855, was entirely lost in a clinical point of view.

The clinical history of circumscribed sclerosis begins with the work of Frerichs (1849), which was very greatly enlarged and perfected by Valentiner (1856). A few years later, the pathological anatomy of the disease was greatly cleared up by the labors of Rindfleisch (1863), Leyden (1863), and Zenker (1865). The French works, which followed those of the above-named investigators, have, however, undoubtedly contributed most to our knowledge of the disease. They defined more sharply the morbid picture presented by the affection, gave due prominence to its essential points, and secured for it a place in pathology as a legitimate and recognizable disease. After the appearance of the first works of Vulpian and Charcot (1866–1868), the latter in particular contributed very greatly to the enlargement of our knowledge of multiple sclerosis; with the help of his pupils (Ordenstein, 1867, Bourneville and Guérard, 1869) he has studied it in an exhaustive manner, from almost every point of view.

About the same time there appeared in Germany an excellent account of a case observed by Leo, which was the first of a long list of careful and full reports (by Schuele, Leube, Ebstein, Kelp, Buchwald, Otto, Berlin, Engesser, Baerwinkel, Hirsch, Westphal, Jolly, Chvostek, and others). These reports have contributed not a little to the enlargement of our knowledge and to the elucidation of different important questions. In France, also, numerous isolated observations have been recorded since the above date (by Liouville, Joffroy, Magnan, and others). In England, however, until the appearance of the recent work of Moxon (1875), the disease was overlooked and almost entirely unknown.

Through the labors of the above-named writers, we have acquired a pretty accurate knowledge of the affection, especially in its practical relations, although numerous questions of detail, some of them not unimportant, are still in an unsettled state, while in the domain of therapeutics in particular almost everything still remains to be done.

Definition.—Multiple sclerosis is a form of chronic myelitis and encephalitis.

It is characterized, *anatomically*, by the development of numerous, insulated sclerotic nodules, varying in size, and of a chronic inflammatory nature, which are scattered irregularly throughout the entire cord, and usually also throughout the entire brain, but which seem to possess, nevertheless, certain spots of predilection. Sometimes a less intense, but more diffuse sclerosis unites the different nodules with one another.

Clinically the manifestations of the disease are very variable and manifold, assuming almost protean shapes. This is the natural result of the development of the nodules in so many different localities, which are seemingly determined by accident.

Motor pareses and paralyses are never wanting, while disturbances of sensibility belong to the less constant symptoms. Disturbances of co-ordination (ataxia) are very frequently observed, and a peculiar, exceedingly characteristic tremor, which accompanies voluntary movements and progressively increases, is almost regularly present. These symptoms are accompanied by a more or less extensive list of bulbar and cerebral symptoms, the most prominent of which are a peculiar disturbance of speech (scanning, monotony of voice), noisy inspiration, impairments of sight, nystagmus, pronounced psychical disturbances of varying degrees, and apoplectiform attacks.

We have to deal here with an affection that runs an exceedingly chronic course—a course moreover which always goes on progressively from bad to worse. Very considerable fluctuations in the severity of the symptoms do occur, it is true, but a permanent improvement is rarely observed.

For practical reasons we shall discuss together the multiple scleroses of the cord and of the brain. The exclusively spinal form of the affection is rare, and the characteristic morbid picture belongs essentially only to the cerebro-spinal form.

Etiology and Pathogenesis.

Hereditary influences may in many cases be regarded as *predisposing* to multiple sclerosis. Duchenne observed hereditary transmission of the disease in one case, and I have myself observed the same thing. Frerichs saw a brother and sister who were suffering from the disease, and a similar case has come under my own observation. The general neuropathic tendency may perhaps play its well-known rôle here also, as hysteria and other nervous disturbances sometimes, though relatively seldom, precede the affection. In other respects but little is known concerning the influence of this diathesis.

According to Charcot's statements, the female sex presents a considerably greater predisposition to the disease than the male sex. The fact that his observations were partly conducted at the Salpetrière (Women's Hospital), will explain the discrepancy between his statements and those of other observers. Chvostek observed sixteen cases, of which ten were men and six women. Of the nine cases observed by myself, four were men and five women. Hence there is no marked difference between the two sexes in this respect.

It is certain, however, that the affection is pre-eminently a disease of youth and middle age. It most frequently makes its appearance during the second and third decades of life; it rarely or never appears after the forty-fifth year, and relatively few cases have been known to occur in children under ten years of age.

Among the *direct causes*, we must mention first, "*catching cold*," the influence of which has been repeatedly demonstrated. The disease has been known to develop after sudden and severe exposure (*e. g.*, a fall into cold water or the like), as well as after continued or repeated exposure to injurious influences, especially to the prolonged action of damp cold. It is impossible to conceal the fact, that the mode of action of these influences is as yet enveloped in the greatest obscurity.

Excessive mental and bodily exertions are also asserted to be exciting causes. It is possible, that they prove so only in already predisposed individuals. In this category belongs also the action of *intense emotions*, particularly of long-continued depressing emotions, such as sorrow, care, etc. These are often advanced as the causes of the disease.

In one of my cases, that of a young lady whose father in all probability died from the same affection, the disease was brought on by fright combined with fatigue and exposure, which were incidental to a fire that broke out suddenly. Hence, in this case, there were a number of injurious agencies active at the same time.

Traumatic influences (injury of the head, concussion of the entire body, railroad injuries, etc.) seem to be decidedly potential. In the etiology of the observations recorded in medical literature, the occurrence of such injuries, some of which, it is true, were received years before the breaking out of the disease, is so frequently mentioned, that it is impossible to assume a purely accidental coincidence.

Pregnancy has also been adduced as an etiological agent (Guérard). Mollière believes that the disease is often developed as a sequel to long-continued and severe *hysteria*. There is no doubt that disseminated sclerosis can be developed as a sequel to *acute diseases*. Ebstein and Westphal saw it after typhus, and Charcot after cholera. Westphal has described some cases of disease following variola, which in all probability must be classed as disseminated sclerosis, although some important features of the disease were wanting in the clinical picture. This theory finds support in the fact, that he subsequently demonstrated the existence of a disseminated myelitis in some similar but more acute cases. Otto has described a similar case.

None of these isolated facts, however, have as yet furnished the slightest information concerning the more delicate processes in the pathogenesis of this remarkable affection.

Pathological Anatomy.

Multiple sclerosis is, as a rule, very readily recognized by the *naked eye*. In a few isolated cases, however—Bourneville has described one such—the cord and brain appear normal, and the existence of nodules of sclerosis is only revealed by microscopic examination.

The alteration appears in more or less *numerous spots and nodules*, which are not confined to the cord, but are scattered in greater or less numbers through the medulla oblongata, the pons Varolii, the cerebrum, and the cerebellum.

The individual nodules are usually recognized easily by their color and consistency. When they lie close to the surface, they often glimmer distinctly through the pia, and in very intense cases, the entire surface of the cord may appear to be studded with grayish and usually sharply defined spots. These are often slightly swollen and elevated above the level of the surface of the cord; more rarely they are somewhat sunken and atrophic, while frequently they are level with the surface, and have consequently undergone no change in volume.

They are usually seen much more distinctly in the cross-sections. More or less extensive spots are seen in every section in the white or in the gray substance, or in both together. These spots are gray or grayish yellow, and when exposed to the air often take on a light reddish tint (like salmon-flesh). They are somewhat *translucent*, resembling jelly or cartilage, or perhaps more opaque; fine white lines are often seen running through them (vessels, fatty spots). They are more or less *sharply circumscribed;* as a rule, the edges are pretty sharply defined against the surrounding healthy tissue, but sometimes they are blurred, and the transition from the diseased to the healthy tissue is more gradual. They are frequently very closely packed, and sometimes become confluent. In form they are rounded, elliptical or more or less irregular, so that it is impossible to describe them very accurately.

The *consistency* of these nodules is, as a rule, considerably increased. They are tough, dense, leathery or cartilaginous, oppose considerable resistance to the cutting instrument, and can usually be recognized by the palpating finger as firm nodules imbedded in the soft nervous mass. In rare instances the nodules are soft, semifluid, gelatinous; these are probably nodules of recent date.

Their *cut surface* is smooth, and uniformly glossy, and gives exit to a very small quantity of clear fluid. In *size* they vary greatly. They may be so minute that they are only revealed by the microscope, or they may be as large as a hemp-seed, a bean or a hazel-nut, or even larger; between the two extremes every possible intermediate stage may exist. In the brain the diseased spots are sometimes very large and diffuse; in the cord they are sometimes greatly elongated (from two to ten centimetres or even longer—three-quarters of an inch to four inches), though confined to single white columns.

Their number varies within the widest limits in the individual cases. Sometimes only a few are found, and again they are so numerous that they might be numbered by hundreds.

The *distribution of the nodules in the spinal cord* is very dissimilar in different cases, and apparently depends entirely on accident. They may be situated in the white as well as in the gray substance. All the white columns may be attacked without distinction; the furrows on the cord do not constitute barriers to the pathological process. In one cross-section the nodules will lie chiefly in the lateral column, in another more in the posterior columns, and in still another perhaps more in the gray substance; in one they will take up the greater part of the sec-
nd in another only a very small part of it; here they will
ned to one side, and there they will involve continuously
des of the cord, etc. (See Fig. 6.)

opinion held by many authors, that the anterior columns are pre-eminently affected, holds true at all events only for a small number of the cases. The more recent German observations include, as it happens, several cases in which the disease attacked chiefly the posterior columns.

Furthermore, numerous nodules are usually found also in the various parts of the caudex cerebri, in the medulla oblongata, the floor of the fourth ventricle, the pons, and the pedunculi cerebri. These parts, together with the walls of the lateral ventricles, the white substance of the cerebral hemispheres, and the white columns of the spinal cord, seem to be the localities for which the nodules show most predilection. The nodules may be scattered throughout the entire extent of the structures forming the caudex cerebri; they are of varying sizes, and are sometimes superficial and again deeply seated, sometimes numerous and closely packed and in other cases more isolated (see Fig. 7). It is very rare for these parts to be found free from disease.

The *cerebrum* usually contains a large number of disseminated nodules, which are very distinctly seen on every section through the organ. The gray substance of the cortex is, however, generally spared by the disease, nodules being only exceptionally found in it. In the central, white substance, the walls of the ventricles, the corpus callosum, etc., they are, on the other hand, exceedingly numerous. As a rule, some nodules are also found in the corpora striata and the thalami optici.

In the *cerebellum*, on the contrary, the nodules are usually few in number. Here, too, they are located by preference in the central white substance, the cortical layers being usually left exempt.

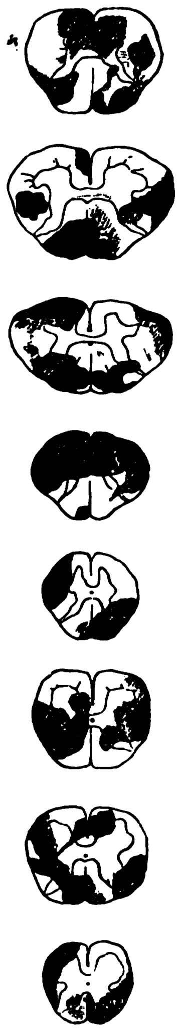

FIG. 6. Semi-diagrammatic representation of the changes in multiple sclerosis, as seen in sections made at different levels of the cord. The dark spots represent the sclerotic nodules.

The account just given applies almost solely to nodules of old standing, such as are met with in the bodies of persons who have suffered for many years from the disease. In these cases we usually find the firm, dense, gray masses, on which the classical picture of multiple sclerosis is based. For an accurate description of the characteristics of the younger and youngest nodules, we must look to the future. Zenker found in one case rounded translucent masses of a soft gelatinous consistency, which presented a homogeneous, structureless and colorless matrix enclosing numerous cells, and which he held to be young nodules of sclerosis. In general, most observers manifest a tendency to regard the gray nodules, which possess a softer, and perhaps semi-fluid consistency, as the younger, and those which are harder and denser as the older. This may be on the whole correct, but at the same time the possibility of individual differences cannot be entirely excluded.

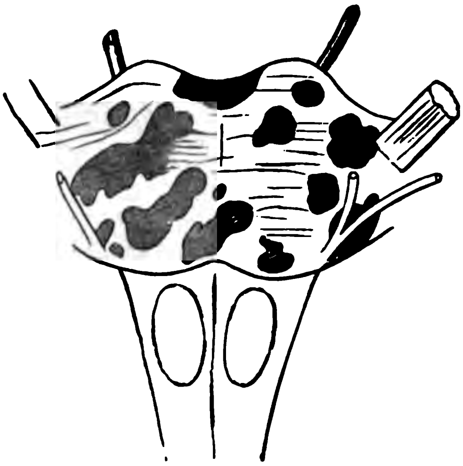

Fig. 7. Distribution of sclerotic nodules on the surface of the pons. After *Leube*, Arch. f. klin. Med. VIII.

Furthermore, it must also be mentioned, that the sclerotic nodules are not by any means limited to the central organs; on the contrary, they not unfrequently extend beyond them and establish themselves in the *nerve-roots* and *nerve-trunks*. This has most frequently been observed in the cerebral nerves. Here, just as in the central organ itself, gray translucent, circumscribed nodules, involving the entire thickness of the nerves, are not unfrequently met with; sometimes several of them are found on the same nerve. Such nodules have been found in the optici, the olfactorii and the trigemini; also in the nerves of the muscles of the eye, in the hypoglossi, etc. On the roots of the spinal nerves, too, such nodules have been repeatedly seen. On the peripheral nerves, however, their occurrence has not yet been demonstrated with sufficient positiveness.

Finally, we have still to mention the fact, that in addition to the disseminated sclerosis, a more diffuse sclerosis has also been occasionally met with in the cord as well as in the brain (*e. g.*, the cases of Schuele, Kelp, Buchwald). The individual, apparently isolated nodules are then united with one another by a more or less altered intermediate tissue. It is probable, that a gradual transition to a truly diffuse sclerosis may take place in this way.

With respect to the *microscopical appearances* of the sclerotic nodules, we may, for all the essential points, refer to the description given of the microscopical changes in chronic myelitis. In the opinion of almost all observers, multiple sclerosis presents the type of *interstitial* chronic myelitis. We shall confine ourselves here to a brief sketch of the histological appearances.

It may be premised, that the sharp line of division between the healthy and diseased tissues, which is apparent when the specimen is examined with the naked eye, cannot usually be recognized under the microscope. On the contrary, a very gradual transition from the normal into the morbid tissue can, as a rule, be demonstrated.

In the nodules themselves we find: more or less marked thickening of the trabeculæ of the neuroglia, swelling and proliferation of the nuclei, large and very sharply defined spider-cells; gradual transition into a fibrillated and delicately fibrous connective tissue; finally, nothing but parallel bundles of fibrils placed close to one another; between them remains of the medulla, fat- and granule-cells and masses of the same substance, free fat.

In the *nerve-fibres* atrophy of the medullary sheath and swelling of a few of the axis-cylinders, most of which, however, remain unchanged; disappearance of the medullary sheaths, persistence of the naked axis-cylinders, which are closely embraced by the small-meshed fibrillated tissue; at last, the axis-cylinders also may become sclerotic, glossy, brittle. Finally, these too disappear in great part, and there remains only the dense fibrillated connective tissue, which contains large and beautiful spider-cells, a moderate abundance of nuclei and a varying number of corpora amylacea; free fat and drops of myeline are then usually wanting.

The remarkably long persistence of the axis-cylinder in the sclerotic tissue, which was pointed out by Charcot and confirmed by Leyden, does not really possess the importance ascribed to it, in the differential diagnosis from other forms of chronic myelitis. At all events, even in advanced cases of sclerosis of the posterior columns (tabes), we not unfrequently find very numerous well-preserved nerve-fibres and axis-cylinders; hence I do not think that the persistence of the axis-cylinders can be regarded as a radical point of difference or an essential characteristic.

A considerable sclerosis of the walls of the *vessels* is regularly observed. They are thickened, blended with the surrounding fibrillated tissue, and very rich in nuclei; not unfrequently they are in a state of pronounced fatty degeneration, and there is an abundant infiltration of fat into the lymph-channels which envelop them. The lumen of the vessel is thereby greatly diminished.

If the sclerotic process extends into the gray substance, the ganglion-cells located therein generally become atrophied. They frequently acquire a strikingly yellow color (yellow degeneration), and are no longer stained perfectly by carmine; later on they undergo colloid atrophy, lose their processes, and may finally be completely destroyed.

Very little attention has hitherto been paid, in cases of multiple sclerosis, to the familiar *secondary degenerations*. I find them hardly ever mentioned, and still, in view of the position and extent of many sclerotic nodules, their absence would seem decidedly remarkable. They seem at all events to occur relatively very seldom; in many cases, however, it might be difficult, without a very searching examination, to decide exactly what belongs to the primary sclerotic nodule and what to the secondary degeneration. A closer study of this question would be desirable.

Jolly's case is the only one in which the descending degeneration of the lateral columns is expressly mentioned. It is not positively certain, however, that the case

belongs in the present category. The cord was free, and the changes in the brain were of a more diffuse character.

In addition to the changes in the central organs themselves, some other subordinate alterations take place, which require only a brief mention. The skull-cap is frequently found abnormal; it may be thickened or thinned, presenting sclerotic or eroded spots. The membranes of the brain and cord are frequently perfectly normal, but in other cases present more or less pronounced evidences of hyperæmia and chronic inflammation. The cerebro-spinal fluid is, as a rule, increased, frequently cloudy, somewhat flocculent, etc.; the ventricles are considerably dilated.

In the other organs of the body (muscles, peripheral nerves, skin, bones, bladder, kidneys, lungs, etc.) the changes already spoken of in connection with chronic myelitis are occasionally observed. Some of them are direct consequences of the severe spinal affection (such as bed-sores, vesical diphtheria, pyelonephritis, etc.), while others constitute more or less accidental complications or terminal diseases (pneumonia, pleuritis, pneumophthisis, typhus, etc., etc.). It is unnecessary to speak of them here.

Pathology of Multiple Sclerosis.

Symptoms.

General clinical history.—In the majority of the cases the disease develops gradually and insidiously. Sometimes, however, it commences abruptly: the scene is opened by an apoplectiform attack, a sudden paralysis, severe headache and vertigo, or the like.

In the cases which develop slowly the initial symptoms are usually very obscure, vague and manifold. Sometimes they are chiefly referable to the spine and sometimes to the brain. In one case there may be slight disturbances of sensation, paræsthesia, weakness, paresis of the lower extremities, uncertainty of gait, disturbances of co-ordination recalling those of ataxia, difficulty in writing, cardialgic attacks (so-called crises gastriques) with vomiting, etc. In another there may be vertigo, headache, staggering gait, tremor of certain muscles or limbs, impairments of speech, of sight and of particular cerebral nerves, psychical disturbances, and a convulsive or apoplectiform attack. The commencement of the disease is very frequently marked by *headache, dizziness, uncertain gait and slight psychical depression.* It is evident, however, that the initial symptoms may occasionally show themselves in all possible parts of the body.

The symptoms slowly become more severe, and new ones are gradually or abruptly added. The disease continues for years with slight fluctuations and occasional, deceptive improvement, but the practised eye can usually recognize a slow, progressive aggravation. The forms in which the disease can present itself are exceedingly numerous, and Charcot was right when he called it *par excellence* a polymorphous affection.

In marked and typical cases the following morbid picture, or something very like it, is developed after the lapse of a variable period. The patients complain of *sensory disturbances which vary* very greatly in intensity, quality and localization. Usually there are only paræsthesiæ; severe pains are less common, but still they do occur. They assume the most manifold shapes, and may present themselves in any part of the body; they may take the form of facial neuralgia, or of girdle-pains, or of lancinating or diffuse pains in the extremities or the trunk, etc. To these are added later, anæsthesiæ of varying degree and location, impairment of the muscular sense, etc. It is a striking fact, however, that in a relatively large number of the cases, these sensory disturbances are very slightly developed or even entirely wanting; at least the objective examination often fails entirely to reveal any of them.

The *motor disturbances*, on the other hand, are generally much more constant and more severe. The first to attract attention are usually pareses and paralyses, which almost always begin in one leg, and extend subsequently to the other leg and then to the arms; occasionally, however, they occur in some other order of development, all imaginable variations from the above sequence being possible. From a simple difficulty in walking, which is usually of a spastic character (see Part I., p. 97), the disease passes slowly through all the stages of development up to complete paraplegia. Muscular contractions (Muskelspannungen) usually make their appearance early, and in the later stages very marked contractures of the lower extremities are the rule; the legs are held as rigid as sticks in a position of extension and adduction. In the upper extremities the paralyses rarely attain such a high grade as in the lower extremities, and contractures are rarer; still both contractures and marked paralyses occur in them.

In a certain number of the cases, there is also true and pronounced *ataxia* of the extremities. The most trustworthy observers report that they have seen exquisitely jerking movements of the legs, setting down of the heels, etc., with the consequent uncertainty in walking and standing; also corresponding ataxic disturbances of movement in the hands, etc.

A particularly striking, and in most cases exceedingly important and characteristic symptom, is an active, *shaking tremor*, which occurs chiefly or exclusively during voluntary movements (volitional trembling), and disappears or becomes scarcely noticeable during rest. As long as the patient remains quietly seated, there is either no tremor at all, or at most a slight shaking movement of the head, or a slight oscillation of the trunk. As soon, however, as he attempts to seize anything with the hand, the tremor sets in; it is a forcible, rhythmical, shaking movement, which increases in violence with the increase in the force of will exerted, and which is very different in character from the irregular, impulsive movements of ataxia, although it is occasionally combined with them. The intended movement is generally carried out correctly, but it is attended by oscillatory vibrations with more or less regular excursions. When the patient attempts to raise a glass to his mouth, he cannot do it without spilling the contents, which are often scattered widely about him; if he succeed finally in the attempt, the tremor makes the glass rattle against his teeth in a rhythmical manner. The oscillations increase as the goal is approached, in a direct ratio with the increasing intensity of the will-force, and the augmenting strain on the attention. The oscillations of the head and the trunk also increase in intensity, when movements of the arms are attempted. When the patient rises and attempts to walk, the tremor involves the entire body. He staggers on his feet, and his entire body, head and arms are shaken by a violent tremor; in the severer grades of the affection he is often unable even to stand, let alone to walk.

As soon as the effort of will is relaxed and the limbs are supported, the tremor ceases. While the patient is in the recumbent posture with the head well supported, no trace of it can, as a rule, be detected, but the slightest effort of the will, any strong emotion, and any excitement will immediately call it forth in a very marked degree. A continuous tremor, persisting also during rest, and hence resembling somewhat the tremor of paralysis agitans, has only been observed in a few isolated cases; in some of these it was a transitory and in others a persistent symptom.

In connection with all these motor disturbances, the condition of the *reflex activity* may vary. Usually it remains unaffected for a long time, sometimes it is diminished, but frequently, especially in the ordinary cases which present paraplegia and muscular contractions, it is increased, the tendon-reflexes in particular being exquisitely developed.

The *vesical* disturbances are usually remarkably trivial, perhaps on account of the relatively slight implication of the gray substance of the cord. In some cases, however, all imaginable disturbances of the vesical function, from the most trivial up to the most severe, viz., retention and incontinence, have been observed, just as in the other forms of chronic myelitis. Usually, however, these symptoms belong to the latest stages. The same statements may be made with respect to the *rectal function* and the *sexual powers*. The latter often remain unaffected for a remarkably long time, but towards the last they almost invariably become impaired, and are finally extinguished.

Trophic disturbances are usually wanting for a long time. As a rule, the blooming appearance of the patients presents a striking contrast to the complete helplessness to which they are condemned by the motor disturbances. Sooner or later, however, but in general only in the last stage, trophic disturbances of various kinds are wont to occur. The muscles become atrophied and lose their electric excitability. Bed-sores are developed, the state of the general nutrition sinks along with the digestive powers, and fever hastens the advance of the cachexia.

The symptoms thus far enumerated are supplemented in most cases by a series of phenomena, whose origin can positively be referred to changes in the brain (*cephalic or cerebral symptoms*). We must mention first certain bulbar symptons, which are among the most important of the entire morbid picture ; first of all, a remarkable *alteration of speech and of the voice.* The speech is slow, hesitating, distinctly scanning, and later on more or less indistinct. The voice is weak, monotonous, and has less staying power. The acts of laughing and crying are accompanied by peculiar, noisy inspirations. Later on the movements of the tongue and lips are not unfrequently impaired, mastication and deglutition are accomplished with great difficulty, etc.

The disorders of the organ of vision are, as a rule, very significant. Temporary or permanent *diplopia*, due to pareses of the ocular muscles, has been observed. A marked *nystagmus*, which, according to the statements of different observers, is increased by any exertion of the will or by any violent emotion, is very common. Finally, *amblyopia* is not infrequent, but it is only in a few cases that it increases to complete *amaurosis*, with atrophy of the optic nerve as its basis.

Furthermore, *psychical derangements* are very generally present. Often, these consist only in slight irritability, changes in disposition, a more active reaction to all sorts of impressions, a great tendency to weep, or to laugh without motive, etc. Impairment of the memory and the intelligence is common, as is also the development of pronounced psychoses; melancholia, monomania taking the form of persecution or of exaltation, finally craziness and complete dementia.

The *attacks of vertigo*, *the sleeplessness and violent headaches*, which are not unfrequently observed, complete this picture; and when we add that in some cases repeated *apoplectiform attacks*, accompanied by high fever and followed by temporary hemiplegia, are observed, we have therewith put the finishing touch to the general description of the clinical history.

This description, however, applies only to the very pronounced and typical cases of the disease. It cannot be denied that the variations are exceedingly numerous; that, on the one hand, this or that symptom may be wanting, while on the other hand, symptoms may occupy the foreground, which will stamp the clinical history with a great resemblance to that of other known diseases. The accidental localization of the principal nodules affords a ready explanation of the fact, that certain cases present an unmistakable resemblance to sclerosis of the posterior or of the lateral columns, or to myelitis transversalis, etc. The inconstancy of often the most important symptoms, the absence of some, the occurrence of others, and the great changeableness of the clinical picture, also find a natural explanation in the great differences in the number and localization of the nodules in the different cases.

An attempt has been made to differentiate different forms of the affection, according to the predominating localization of the disease. The most frequent form has been accordingly designated as the *cerebro-spinal*, because in it the nodules are found both in the brain and the cord; in contrast to it, a purely *spinal* form (with exclusive spinal localization) and a purely *cerebral* form (with exclusive cerebral localization) have also been set up. We shall return to this point in another place, and we shall see that in practice this sharp differentiation is, as a rule, impossible.

The course of the disease is usually exceedingly slow and sluggish. A very striking improvement sometimes occurs—an actual remission that almost simulates a cure. It is wont to be deceptive, however, and is rarely persistent. Usually the disease makes continuous, though often very slow progress. Its duration must be reckoned by years, often by many years. As time elapses, all the symptoms increase in intensity and extent; the paralysis, the contractures, and the psychical weakness assume more and more the upper hand, and bring about an exceedingly deplorable state of affairs. The speech becomes more and more unintelligible, the pains augment, and the restlessness of the limbs becomes unbearable; affections of the bladder and bed-sores, with all their consequences, are superadded. Those patients are most fortunate who are rapidly carried off by some acute intercurrent disease (pneumonia, septicæmia, typhus, cholera, etc.), as they thereby escape a lingering death after unutterable sufferings, from the advancing general cachexia.

Analysis of the Individual Symptoms.

It is not necessary here to go into all the details, since a great number of the phenomena met with are to be explained exactly in the same way, as the same symptoms in chronic myelitis. Hence, we can, for many of them, refer to the preceding chapter. This applies especially to the disturbances of sensibility, the paretic and paralytic phenomena, the state of the reflex activity, the muscular contractions and contractures, the vesical symptoms, the occasional atrophy of the muscles, the bed-sores, etc. Of course a part of these symptoms may be of cerebral origin, *e. g.*, the pareses and paralyses, which may be due to sclerotic nodules situated in the thalami optici, the corpora striata, the pedunculi, etc.

Many authors speak of the *absence* of marked, objective disturbances of sensibility, which they claim to be especially characteristic of the disease. This is, however, decidedly incorrect. Berlin found disturbances of sensibility noted in fifteen of the thirty-nine cases examined by him. In not a few cases all possible varieties and grades of sensory disturbance are found. In Engesser's case, there were distinct diminution of the cutaneous sensibility and considerable impairment of the muscular sense, with much staggering when the eyes were closed; but without ataxia. Other cases have presented marked ataxia, without any disturbance of sensibility, and also without any staggering when the eyes were closed—a fresh proof that this last symptom has a direct dependence solely on the sensory disturbance, and has no connection with the ataxia as such. It is certainly remarkable, that in many cases the disturbance of sensibility should be so exceedingly trivial, or even entirely absent, in spite of widespread multiple sclerosis involving also the posterior columns. Schuele has investigated this question thoroughly, and found in the immunity from the lesion of certain parts of the posterior columns and the gray substance, the conditions which in all probability secure the undisturbed conduction of the sensory impressions, despite apparently large gaps in the normal sensory tracks.

We must now devote some space to a special discussion of that very important and characteristic symptom, the *shaking tremor during voluntary movements* (the volitional trembling—Intentionszittern—of F. Schultze). This presents a very peculiar and easily recognized *ensemble*. While the patients remain seated, we notice only a slight rhythmical trembling and oscillation of the trunk and the head, the extremities remaining quiet. It must be borne in mind in this connection, that in the act of sitting the muscles of the trunk are constantly in a state of slight action, which is requisite for the retention of the equipoise.

As soon as any movement is made with the upper extremities, an active trembling and shaking becomes noticeable in them, while at the same time the oscillations of the head increase in intensity. All delicate and even all coarse manipulations are thereby impeded or made impossible; in writing, the letters are very greatly distorted, and finally become entirely undecipherable, etc.

When the patients attempt to stand and walk, the entire body is violently agitated, the legs being particularly affected. In the highest grades of the affection, both walking and standing are rendered impossible by this shaking; in the slighter grades, it is observed particularly in changing from one position to another, in rising from a chair, at the commencement of a walk, etc.

On the other hand, when the patients are in a position of perfect rest, lying comfortably in bed, no trace of the tremor can be noticed, at least in the majority of the cases. Strong emotion (anger, irritation, embarrassment, shame) will, however, excite the tremor in these cases even during the state of rest, just as it will notably increase it during voluntary motion. In contrast to the above, it is recorded of isolated cases, that a shaking movement of the extremities was present even during perfect mental and bodily rest, and persisted more or less uninterruptedly for days, weeks, or even longer. I have myself observed one such case. The unfortunate patient was almost constantly tormented in the most pitiable manner by tremor-like, twitching movements of the extremities; there was but one way in which the restless movements could be controlled even for a few minutes, and that was by firmly holding the limbs.

It is very probable that in such severe and intense cases even the automatic movements of voluntary muscles, *e. g.*, the respiratory act, or the half involuntary movements of the muscles of the eye, the muscles of facial expression, etc., are capable of exciting and keeping up the shaking tremor, while the body is apparently at rest.

In the great majority of the cases, however, and in the first stages of the disease, it is always a very essential and characteristic point, that the tremor is ingrafted on the voluntary movements, that *it appears with every innervation of the muscles.* Even in the later stages, when the tremor has become more continuous, it is at all events certain that it is enormously increased by every attempt at movement. As a rule, it is wanting during absolute rest, and appears in connection with all voluntary or involuntary movements, becoming more intense as they become more forcible; the more extensive the movement, the more forcibly it is performed, the more the attention is directed to it, so much the more violent is the tremor.

Therein lies also the *essential point of difference between the tremor of multiple sclerosis and that of paralysis agitans*, two forms of tremor which were almost invariably confounded until Charcot pointed out their exact differential characteristics. In paralysis agitans the trembling is observed also during perfect rest; it is not increased by voluntary movement, but, on the contrary, the patients are able—at all events in the earlier stages of the affection—to control it for a time at least by force of will; any voluntary movement, any change in the position of the limb, is sufficient to still the tremor for a few minutes, but it gradually begins again after the limb has lain quiet for a time. The character of the tremor also is essentially different in the two affections. In paralysis agitans the oscillations are much smaller and more frequent; they possess more of the character of a true tremor, than do the wide oscillations of multiple sclerosis. In the latter, moreover, the tremor of the hands frequently assumes a peculiar and very characteristic type; it looks as if the patients were about to spin, or to mix pills, or the like. The head is seldom or never involved in the tremor of paralysis agitans.

As far as I can judge from my own experience, the trembling of paralysis agitans can in most cases be easily distinguished from the shaking of multiple sclerosis. Any one who has once observed closely and compared the two forms of tremor, will never confound them together in pronounced cases. It would be very wrong, however, to conceal the fact, that cases of multiple sclerosis occur, in which the tremor resembles in character very closely that of paralysis agitans, and in which the decision as to the quality of this one symptom may be very difficult—cases in which the two forms of tremor exist side by side. It is in my opinion much more correct, to assume a combination and complication of two different clinical processes in such cases, than to deduce from them, few in number as they certainly are, the conclusions that the two forms of tremor are not essentially different, and that they can occur promiscuously in both diseases. The former view is supported by the great majority of the observations, which speak plainly in favor of the essential difference of the two forms of tremor. We must wait for more accurate observations, however, before passing finally on the correctness of this view.

With actual *choreic* movements, the shaking tremor of multiple sclerosis cannot be easily confounded in ordinary cases. The movements in chorea are very irregular, violent and impulsive, and not rhythmic or oscillating as those of multiple sclerosis. They occur also during rest very abruptly and without apparent cause. In chorea the direction of an intended movement is altogether distorted ; it is zigzag and irregular: in multiple sclerosis the general direction of the movement is preserved, but the line of movement is wavy, oscillating about a central line. This holds true, at all events, in the typical cases of the two diseases ; here too, however, it seems to me that combinations and undecided intermediate forms occasionally occur, in which doubt may arise as to the proper classification of the existing motor disturbance.

These remarks apply also to true *ataxia*, that disturbance of co-ordination which is present in its typical form in the sclerosis of the posterior columns. Here, too, the differentiation is very easy in ordinary cases. The ataxic movements do not constitute a regular, rhythmical trembling or shaking; on the contrary, they are disorderly, immoderate, jerking movements, which make their appearance at the moment a voluntary impulse is formed and frustrate the intended movement. It is very difficult to describe them in detail, but when once seen and studied, they can scarcely be confounded with anything else. Here too, however, and much more frequently even than in the cases of paralysis agitans and chorea, combinations and mixed forms occur, which permit the development of both motor disturbances in one and the same patient, and which lead uncritical observers to confound them together both in description and in pathogenesis. It is, in my opinion, exactly in these cases, that it is most easy to recognize the combination of *the two* motor disturbances, to demonstrate the simultaneous occurrence of ataxia and volitional tremor; in fact it has already been repeatedly demonstrated by different observers. The pathogenetic explanation of this combination is ready at hand, since the anatomical basis of the ataxia is given in the location of sclerotic nodules in the posterior columns. Other symptoms of sclerosis of the posterior columns are usually present in such cases, viz., lancinating pains, anæsthesia, impairment of the muscular sense, staggering when the eyes are closed, etc. With regard to the volitional tremor itself, we are, it is true, not yet in an equally desirable position, for we are unable to ascribe it even with probability to any specific anatomical localization.

The tremor is one of the most constant, and unquestionably one of the most characteristic symptoms of multiple sclerosis; whenever it is met with, it must direct the attention to that disease. It is not always present, however, not a few cases having been recorded, in which it was absent throughout the entire course of the disease (Leube, Ebstein, Engesser, Jolly, and others), cases, moreover, to which Charcot's suggestion, that it may perhaps have existed during a time when the patients were not under observation, cannot well apply. It is certainly true, that the characteristic tremor diminishes in the later stages of the disease, and finally disappears entirely. This diminution progresses in an inverse ratio to the increase in the prominence of the paralysis and the contractures. Another form of tremor, however, frequently appears at this time, namely, the clonic tremor on passive dorsal flexion of the foot (the so-called epilepsie spinale of Brown-Séquard and Charcot). We have already shown (Part I., p. 101) that this is a reflex phenomenon, and that it is usually connected with increased tendon-reflexes. Beyond the external form, it has nothing in common with the volitional tremor, and only a superficial observer would confound it with the tremor that accompanies voluntary movements, and utilize it in the characterization of multiple sclerosis.

The origin of the typical volitional tremor of multiple sclerosis is as yet exceedingly problematical. It is a variety of derangement of co-ordination, which, however, when closely examined, differs very essentially from true ataxia, and hence must certainly have another anatomical basis. Charcot's theory, that the relatively long persistence of the axis-cylinders in the sclerotic nodules might perhaps have something to do with it, because the impulse of the will would be conducted through the naked axis-cylinders only in a sort of jerking manner, will certainly not find many adherents. It is open to too many objections. The theory, that some specific *localization* of the sclerotic nodules is answerable for the production of the tremor, is certainly more plausible, and it is maintained by many authors. Ordenstein asserts, that it is principally the pons and the parts of the brain situated in front of it, that come into question here, and Hammond says, that the tremor is wanting in the exclusively spinal cases.

In point of fact, if we pass in review the recorded cases of simple chronic myelitis, we do not find this characteristic tremor enumerated among the symptoms, even when several centres of disease exist in the cord. The matter becomes more difficult to decide, when the individual cases of multiple sclerosis are under examination, because in them the wide extension and great multiplicity of the nodules place almost unconquerable obstacles in the way of every attempt to draw a simple and clear conclusion. Still, a critical study of these cases reveals some facts that are worthy of note. Thus Ebstein describes a case of purely spinal localization *without* tremor, but with ataxia; in Kelp's case, on the contrary, which belonged to the purely cerebral form, the tremor was present. Engesser also describes a case *without* tremor, in which the localization was predominantly spinal, and Vulpian's first case was of the same sort. From these facts it seems to follow, that the *localization of single nodules in the brain is, in point of fact, necessary for the production of the tremor.* It is still more difficult to decide exactly what parts of the brain must be affected, in order to produce the tremor. I have examined twenty-two recent cases, with a special view to the decision of this question. In all of them which had presented the tremor during life, the structures of the caudex (pons, medulla oblongata, pedunculi, etc.), besides other portions of the brain, were particularly involved in the sclerosis; on the other hand, in the few cases in which the tremor was absent during life, although nodules existed elsewhere in the brain, there were either none at all or at most only small ones in the pons, the oblongata, the cerebellum, etc. (See the cases of Jolly, Leube and Engesser). Uncertain and ambiguous as these facts may be, they at all events speak in favor of the opinion expressed by Ordenstein, that the localization of the nodules in the pons and the parts of the brain situated anterior to it, is an essential condition for the production of the tremor. This conclusion withal necessarily rests on very insecure foundations, and numerous observations are requisite for its complete confirmation. At all events, the appearance of volitional tremor furnishes even now valuable evidence that a pathological process has been established in the brain itself, evidence which, in doubtful cases, may prove of great diagnostic value.

Among the motor symptoms, the *tonic contraction* of the paralyzed muscles, which appears in the later stages and may subsequently increase to very marked *contracture*, also deserves mention. It affects pre-eminently the lower extremities, which are also, as a rule, more intensely affected by the paralysis. At first isolated attacks of spasmodic extension, of tonic stiffness of the legs, etc., occur, either spontaneously or when the patients attempt to perform some movement; or they may be excited by external cutaneous irritants, or probably also by violent emotions. In a certain stage of the disease, movements, which are otherwise still practicable, may be greatly interfered with by these attacks of stiffness and rigidity. The attacks become progressively more frequent and more prolonged, until finally permanent contractures in the position of extension are developed; the legs are as rigid as sticks, and are held closely together by contracture of the abductors; the feet are in the varo-equinus position. Sometimes, however, contractures in the position of flexion are developed at a later period. These contractures are almost always accompanied by a considerable augmentation of the tendon-reflexes. The already repeatedly described, clonic twitching of one or even of both legs excited by passive dorsal flexion of the foot (called by Charcot tonic spinal epilepsy), has been mentioned particularly by many observers. This clonus can be excited by cutaneous irritation, as well as by passive dorsal flexion of the foot; it is also excited occasionally by voluntary movements and by the act of defecation or of micturition.

The upper extremities are rarely the seat of contractures. Still, exceptions do occur, when the paralysis has extended to the arms; the contractures have then been observed both in the flexed and the extended positions. Schuele observed in one case a temporary, cataleptic or tetanic rigidity of the muscles of the upper extremities.

The explanation of these phenomena is probably the same as that of the analogous symptoms in simple chronic myelitis: interruption of the tracks for controlling the reflex actions and increase of the reflex activity on the one hand, and direct irritation of the motor tracks on the other. Their anatomical basis must, in the present state of our knowledge, be assumed to be the development of sclerotic nodules in the lateral columns and the consecutive, descending secondary degeneration of the same columns.

Among the cerebral disturbances, the *impairment of speech* deserves to be mentioned first; it is a frequent, a striking, and, for the diagnosis, an important symptom. The so-called *scanning speech* is the form of impairment which is most frequently observed. The speech is slow, drawling, and hesitating; every syllable is pronounced separately and distinctly, the words being as it were scanned. Later on, particular letters are pronounced indistinctly and replaced by others, a peculiarity that was especially marked in Radlick's case. Finally, in the more intense degrees, the entire articulate expression becomes indistinct, lalling, and even unintelligible. The impairment consequently is not a true stuttering, and neither is it the form of impediment which is so common in progressive cerebral paralysis. In paralysis, the words tumble over one another, syllables and words are left out entirely or are jumbled together in a confused mass, and letters and syllables are inserted in the wrong places; in multiple sclerosis, on the other hand, the words fall from the lips in a hesitating, regular, scanning manner.

The mobility of the lips is often impaired, and they may present slight twitching movements in speaking. In many cases the tongue can only be put out in a slow and tremulous manner.

The *monotony of the voice*, which has been already spoken of, is in most of the cases a striking peculiarity. The voice remains in the same key, is weak and low, and often almost whispering; it breaks readily when forced efforts are made. Leube examined one such case with the laryngoscope, and found that the vocal cords could be brought together, but that their tension was soon relaxed and frequently changed. In one case observed by myself, the monotony alternated with the exactly opposite condition; there was a constant, rhythmical, perfectly regular change in the key at fixed musical intervals, *e. g.*, a third, so that the key was altered in the pronunciation of every syllable, always observing, however, the same interval, just as if the patient were constantly singing a-c, a-c, a-c, etc. The effect produced was very peculiar.

Leube's observations show, that this peculiar disturbance of the voice probably depends on a diminution in the innervation and the capacity of the vocal cords. Whether the scanning is due to the same cause is still questionable. Leube attempts to explain it also by the exhaustion and weakness of the cords; their capacity, according to him, is only equal to the production of isolated syllables, and hence the syllables are enunciated in an isolated manner. Under such circumstances, however, we should rather expect to find the articulation confused and precipitate. Persons who suffer from weakness of the vocal cords due to other causes are not wont to scan. It seems to us, consequently, that in order to account for the scanning, we must assume the existence of disturbances in the nerve-tracks that preside over articulation.

The entire disturbance of speech can in most cases be referred to the presence of sclerotic nodules in the medulla oblongata and the pons. Still we cannot exclude the possibility, that nodules located further forward in the nerve-tracks for the organs of speech may be answerable for this disturbance. Jolly's case certainly presented the impairment of speech, and still there were absolutely no changes in the pons, and only doubtful alterations in the medulla oblongata.

In connection with the above we must consider the *manifestations of bulbar paralysis*, which have been observed in several cases (Joffroy, Leube, Schuele, Jolly, and others), and which give us a distinct glimpse of the typical form of that affection. Impaired mobility of the lips and tongue, difficulty of swallowing, paralysis of the velum palati, permanently open mouth, increased secretion of saliva, etc., are the symptoms. In the majority of the cases they are certainly due to the presence of nodules in the bulb, but Jolly's case proves that this is not invariably and necessarily the case; in it the morbid process a wide-spread sclerosis, was located more centrally.

The *disturbances of respiration*, which are often observed, also belong here beyond a doubt. Especially characteristic are the disturbances in the innervation of the larynx, which manifest themselves on the one side by the already described monotony of speech, and on the other by the *stridulous inspirations*, which are frequently observed during laughing, weeping, etc. They may probably be explained by a paresis of the crico-arytænoidei postici.

The *nystagmus* is usually the most striking of all the eye symptoms. It consists of short twitches, which have a horizontal direction, and draw the eyes sometimes in an outward and sometimes in an inward direction; they are at times persistent, but again may occur only during forced accommodation, or when movements are performed with the extremities. In the latter case the eyes present nothing abnormal, when the body is in a state of general rest. This symptom is pretty common; according to Charcot it is met with in about half of the cases. Whether, and to what extent, this nystagmus differs from the ordinary nystagmus of eye-patients; whether or not it is an essentially different disturbance, are questions that must still be left open. It is probable that, in many cases, we have to deal with the "ataxic nystagmus" (disturbance of co-ordination of the muscles of the eye), recently differentiated and described by Friedreich;[1] in other cases it may be analogous to the volitional tremor.

Both physiological and pathological facts (compare the account given in the work of Friedreich just cited) warrant the assumption, that the cerebellum, the corpora quadrigemina and the medulla oblongata possess important functions relating to the co-ordination of the movements of the eyes. Hence it is probable, that the presence of sclerotic nodules in these structures may be the cause of the nystagmus in multiple sclerosis. This still lacks positive demonstration, however.

The next symptom in point of frequency is *amblyopia*, a progressive weakness of sight, sometimes accompanied by color-blindness and diminution of the field of vision, and, in rare cases, increasing to complete amaurosis. Sometimes the development of the amblyopia is preceded by photopsia, flashes of light, etc. On ophthalmoscopic examination the papilla is sometimes found perfectly normal or only partially diseased, while in other cases it is in a state of pronounced white atrophy. All this is probably produced chiefly by sclerotic nodules in the tractus and the nervi optici. The fact that these nodules are essentially of an interstitial character, affecting the nerve-fibres only at a later stage, and in a secondary manner, is, according to Charcot, the reason why the amblyopia is not very intense and complete; the axis-cylinders, being left intact for a long time, are still capable of performing their functions.[2]

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[1] Ueber Ataxie mit besonderer Berücksichtigung der hereditären Formen. Virch. Arch. Bd. 68. 1876.

[2] See also *Foerster*, Beziehung der Krankheiten des Nervensystems zum Sehorgan,

Finally, *diplopia* is not unfrequently observed, sometimes as an initial and transient symptom, sometimes in the later stages, when it depends on complete and permanent paralysis of the different muscles of the eye. There can be no doubt that these later disturbances are to be ascribed sometimes to nodules in the peripheral nerves of the ocular muscles, sometimes to nodules in the intra-cerebral tracks for the innervation of these muscles.

Impairments of *smell, taste, and hearing* occur in isolated cases, but they are rare, and hence possess no great importance.

Psychical disturbances, on the other hand, are common, and belong essentially to the morbid picture. In the commencement and in the milder cases, simple depression of spirits and impairments of the memory, the intelligence, and the intellectual capacity, are observed. With these are combined attacks of yawning or sobbing, and a great tendency to laugh or weep without motive. Later on the patients become stupid, the face acquiring a correspondingly apathetic and varyingly stupid expression. Sometimes, however, the mental disorder becomes very pronounced; an intense melancholia, sometimes accompanied by stupor, rejection of food, etc., or mania with delusions of persecution or of grandeur, conditions of exaltation, etc., may be developed. These disorders can run their usual course, terminating finally in complete destruction of the intellectual life.

It is clear, that all these disturbances can be ascribed only to the development of nodules of sclerosis in the hemispheres of the cerebrum. It is scarcely necessary to add, however, that we have not yet discovered the exact relations of the specific localizations of the nodules to the particular forms of the mental disorder.

The same may be said with respect to an almost constant, or at all events, in all stages of the disease, a frequent symptom, viz., the *vertigo*. This often makes its appearance very early, even in the prodromal stage. According to Charcot, the dizziness is usually of the turning variety; the patients feel as if they themselves were turning around, or it seems to them that all the

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in the Gräfe-Sämisch Handbuch d. Angenheilk. VII. 1. p. 104; and *Kiesselbach*, Zur Kenntniss der grauen Degener. der Sehnerven bei Erkrankungen des Cerebrospinalsystems. Diss. Erlangen. 1875.

surrounding objects are engaged in a gyratory movement. This vertigo occurs in short, isolated attacks. Its mode of development is entirely unknown. Occasionally an existing diplopia may give rise to a visual vertigo (Gesichtsschwindel) which is easily distinguished from the other form.

We have, last of all, to mention one other group of symptoms, which also stands in direct connection with the brain, viz., the *apoplectiform attacks*. These are apparently entirely analogous to the apoplectiform attacks in progressive paralysis, in which disease they have been long known and accurately studied.

It has not yet been satisfactorily demonstrated that the *epileptiform* attacks, which are likewise not infrequent in paralysis, occur in multiple sclerosis as well. Charcot has not yet met with them. On the other hand, they were observed in Kelp's case, and Leube reports one case (without an autopsy) in which an epileptiform attack was observed.

The apoplectiform attacks occur only in a comparatively small proportion of the cases of multiple sclerosis—about one-fifth of them, according to Charcot. They are characterized by the abrupt development of severe cerebral symptoms, accompanied by considerable elevation of temperature. After slight prodroma, such as a feeling of oppression in the head, an apathy and an obscuration of consciousness appear, which in the course of a few hours may increase to deep coma. The face is red and hot, the pulse quick, and the temperature rises to 40° or 41° C. (104°-105⅘° F.). Very soon the existence of hemiplegia can be demonstrated by the complete relaxation of the extremities of one side. After a shorter or longer interval (one or two days) the consciousness reappears, the temperature falls, and the patient sinks into a deep sleep, from which he awakens relatively well. The hemiplegia, however, persists for a few days longer, and then disappears gradually. These attacks are nevertheless invariably followed by a general aggravation of the disease.

Attacks of this sort may be repeated several times. They may recur every few months, or even at intervals of a year. Sometimes death occurs during the attack, while the patient is in a condition of deep coma.

The interpretation of these attacks is still very obscure. They differ from true apoplexy particularly in the high temperature of the body, the, as a rule, gradual development of the coma, and the rapid disappearance of the hemiplegia.

The autopsies in such cases reveal no signs of acute congestion of the brain. Charcot could never discover in them either œdema or hyperæmia, or, in fact, anything but old changes. He draws attention, however, to the fact, that these attacks are only met with in those cases of the disease, in which the pons and the medulla oblongata are involved either primarily or secondarily. We are, however, still ignorant of the actual cause of these very transient but violent attacks. The suggestion, that they perhaps indicate the acute development of new nodules of sclerosis, has little to support it; they would in that case be much more common.

The above embraces all the chief symptoms of multiple sclerosis, but it still remains for us to mention briefly *a few uncommon symptoms*, which are occasionally met with, although they do not belong to the typical morbid picture. The first of these to be mentioned is the *muscular atrophy*. This may attain a very high grade, and may present itself in any part of the body—in the upper or the lower extremities, in the neck, the face, or even in the tongue (Ebstein). It is unquestionably to be explained by the localization of sclerotic nodules in the corresponding parts of the gray substance.

With this atrophy the condition of the *electric irritability* stands in the most intimate connection. The latter usually remains intact for a long time, or presents at most only slight quantitative changes. In the later stages, when there is increasing atrophy, the electric irritability may diminish very considerably; at such times accurate examination will undoubtedly reveal also the reaction of degeneration. Thus, Leube observed marked impairment of the electric irritability with indications of the reaction of degeneration. In Engesser's case the irritability was at first increased, and subsequently considerably diminished. The customary conclusions with respect to the nutritive state of the muscles and the condition of the gray substance corresponding to them, are to be drawn from these facts. The electrical examination has not, however, up to the present time proved of any great practical value.

The *disturbances of the vesical function* are also variable and inconstant. They are often wanting for a long time, and then set in in various forms and degrees; they are sometimes marked by great fluctuations in intensity (Engesser's case), but do not present anything that is at all characteristic. The entire history of the vesical complications is essentially the same as in chronic myelitis. This may be said also of the affection of the sphincter ani.

The disturbances of the *sexual functions* are also very inconstant. Sometimes the sexual power is retained for a long time, and again it is lost at an early period. In some cases excessive onanism gives evidence of unusual sexual excitement.

In a few cases the so-called *tabic symptoms* are developed in a very pronounced manner; disturbances of the cutaneous sensibility, impairment of the muscular sense, lancinating pains, girdle-sensation, distinct ataxia, staggering when the eyes are closed, etc., are observed in such cases. In most of these cases, this may be explained by the development of a large sclerotic nodule, or of several such nodules, in the posterior columns. A combination of band-shaped sclerosis of the posterior columns with insular sclerosis of the rest of the nervous system, is also possible. Such cases may interpose grave difficulties in the way of a diagnosis. As a rule, however, the presence of other symptoms, which belong only to sclerosis, will clear up the matter. At all events, the cases of hereditary ataxia described by Friedreich (l. c.), which, as far as their clinical history is concerned, occupy a sort of intermediate position between ordinary sclerosis of the posterior columns and insular sclerosis, but still must unquestionably be classed only under sclerosis of the posterior columns, warn us to be very careful in making a diagnosis. We shall return to this subject in another place.

In a similar manner, the symptoms of the so-called *lateral sclerosis* are not unfrequently found in the clinical history; paresis of the lower extremities, muscular contractions, increased tendon-reflexes without disturbances of sensibility, etc. These symptoms may even exist alone for a long time, and their true significance may then only be cleared up by the development of cerebral symptoms at a later period. A primary or secondary sclerosis of the lateral columns accounts sufficiently for this state of affairs, as has already been stated above.

But little progress has as yet been made in the differentiation of *different forms of multiple sclerosis.* The French authors distinguish a *cerebro-spinal*, a purely *cerebral*, and a purely *spinal* form. The correctness of this division has not yet, however, been demonstrated with satisfactory clearness, and it is even positively rejected by some authorities (Buchwald *et al.*). A sharp separation, an unquestionable limitation of the affection to the brain *alone* or to the cord *alone*, is in all probability very rare. Still, there undoubtedly are cases, in which the localization is so predominantly in one or the other of these nervous centres, that we are justified in speaking of a cerebral and a spinal form in a somewhat wider sense.

The cerebro-spinal form has been described in the preceding pages. The *cerebral* form seems to be relatively very rare. The cases recorded by Kelp and Jolly should probably be classed here. In this form only the cerebral symptoms are present; the psychosis usually occupies the foreground in the clinical picture. The tremor is said to precede the paralytic manifestations. In other respects the clinical picture cannot differ greatly from that of the cerebro-spinal form; it is difficult to exclude the simultaneous implication of the spinal cord.

The limitation of the *spinal* form to the cord is usually more easily recognized. Generally, it is sufficiently characterized by the absence of the cerebral symptoms, particularly the nystagmus, the volitional tremor, the psychical disorders, the vertigo, the apoplectiform attacks, etc. The disturbances of speech and of co-ordination may, however, be present. In Ebstein's case, for instance, there were no cerebral symptoms and no tremor, but there were disturbances of speech and of co-ordination. In Engesser's case, which also presented an almost purely spinal type, and formed a sort of transition to diffuse chronic myelitis, the tremor, the disturbance of speech, the nystagmus, the vertigo and the apoplectiform attacks were wanting. Vulpian also de-bes a purely spinal form without tremor, with the exception he reflex clonus in the leg, which, of course, must not be con- nded with the volitional tremor. In cases like these, the ely spinal localization can be easily recognized. The great iculty, however, will be to determine the existence of mul- e nodules. Usually, the clinical picture will present a hope- similarity to that of simple chronic myelitis, and we can y regard it as a happy accident, when the localization of the ules is such, that we can recognize from the symptoms a ultaneous affection of several isolated parts of the cord. A se study of the symptoms will enable us to succeed in this in ne cases. It is unnecessary to enter into further details on s point.

There still remains a certain number of cases, which I might m *unusual or anomalous*, in which the autopsy reveals a ltiple cerebro-spinal sclerosis, although during life the charac- ristic symptoms of the affection were absent, and a diagnosis as consequently impracticable. An example of this is furnished r the case published by Westphal, in the new Charité-Annalen; this case the only symptoms were paraplegia with contractures d coincident dementia. A very similar case was recently ob- rved in the hospital at Leyden, and was published by Killian nder the name of myelitis diffusa. The clinical history of the se is very unsatisfactory (dementia, extensive paralysis with ntractures), but anatomically it was plainly nothing else than disseminated, cerebro-spinal sclerosis. Several cases similar to ese are to be found in medical literature. Here, as well as sewhere in the pathology of the central nervous system, we ust accustom ourselves to the facts, that processes apparently milar in nature and extent do not always produce the same mptoms, and that, as a natural consequence, we shall be unable make an accurate diagnosis during life in not a few cases.

Course—Duration—Terminations.

Charcot has very appropriately distinguished three stages, to which the as a rule exceedingly slow and chronic course of e disease can be divided. His *first stage* embraces the commencement and development of the disease up to the appearance of marked incapacity for motion, with contractures. This stage may last from two to six years or even longer.

Development generally very slow; sometimes begins with cephalic symptoms, headache, vertigo, uncertain gait, etc.; more frequently, however, with spinal symptoms—paresis of the lower extremities, etc.; in the latter case the supervention of cerebral symptoms, the appearance of tremor, etc., are necessary to complete and clear up the clinical picture. In rare cases a more rapid development with more violent manifestations is observed: occurrence of an apoplectiform attack, gastralgic troubles, etc., to which paralyses, disturbances of co-ordination, tremor, etc., are superadded in rapid succession.

Very slowly and gradually the above described syndrome is fully developed. Considerable ameliorations may occur, and sometimes a very striking improvement, which persists for months and years, is observed, but the progressive course of the disease is only temporarily checked. Sometimes sooner, sometimes later, the various characteristic symptoms appear, until finally the complete morbid picture is developed. The patients become more and more helpless, their intellectual powers more and more impaired, complete paraplegia confines them to the bed, the tremor deprives them of the use of their hands, and at last the legs become rigid, contractured, and the seat of frequent spasmodic clonus.

The *second stage*, that of the completely developed disease, is now attained. This stage too can last a number of years, from four to six or even more. The disease remains in general at the same point, without producing disturbances of the general nutrition or other threatening symptoms. In spite of their helplessness and rigidity, in spite of the eternal trembling and th constant confinement to bed, the patients often remain wel nourished, and present a tolerably fair appearance, the slow bu unceasing progress of the affection being often appreciable onl to the practised eye of the physician.

Gradually the *third stage* appears on the scene; it is chara terized by impairment of the vegetative functions, with simultan ous development of threatening nervous symptoms. Loss appetite, digestive disturbances, and emaciation set in; the paralysis of the bladder leads to cystitis; bed-sores with consecutive pyæmic or septicæmic fever are developed; the increasing bulbar symptoms impair the nutrition still more, and apoplectiform attacks threaten life directly.

Under these circumstances death is no longer distant. The patients may succumb before the augmenting difficulties of deglutition and respiration, or in an apoplectiform attack (sometimes with enormous increase of temperature during the agony), or the general cachexia may lead to the gradual extinction of life. Not unfrequently, however, death is brought about more rapidly by some intercurrent disease, such as typhus, pneumonia, pleurisy, pulmonary consumption, or the like. Pulmonary affections, in particular, are among the most frequent complications of the last stages.

The *duration* of the disease is very variable. There are cases which terminate fatally in one or two years, but they are rare. In other cases the disease drags along for ten, twelve, seventeen, or twenty years. The usual duration is about five to ten years.

The *termination* of the disease seems to be invariably death. It is true that marked fluctuations occur as it runs its course, and ameliorations have been observed that seemed to border on a cure, but they were not permanent. No well-authenticated case of recovery has been reported up to the present time. Vulpian records a case of multiple sclerosis, that was influenced in a remarkably favorable manner by an intercurrent varioloid; the symptoms disappeared entirely, but returned again after the lapse of three years. While speaking of chronic myelitis (see p. 449) we mentioned the existence of similar experiences.

Diagnosis.

The recognition of multiple sclerosis may, under some circumstances, be a very easy matter, and under other circumstances be very difficult, or even actually impossible. The purely cerebral form does not properly come into question here; it cannot, moreover, be easily confounded with diseases of the cord. The purely spinal form, as has already been stated, is frequently undistinguishable from simple chronic myelitis; when reduced to its elements, moreover, it is really nothing more than this.

It only remains for us, consequently, to consider the *cerebro-spinal* form, which is, however, by far the most frequent. In pronounced cases it can be recognized with the greatest facility; in the rare, anomalous cases, on the other hand, the recognition is very difficult, or even impossible. The symptoms may be so trifling, so vague and undecided, and so ambiguous, that a diagnosis is either impossible, or can only be made with a certain degree of probability.

When, however, all or the greater part of the following symptoms are present, the disease can be diagnosticated without difficulty: commencement with vertigo and uncertainty of gait; more or less marked paresis and paralysis of the extremities, combined with the characteristic tremor and sometimes also with ataxia, and later on with muscular contractions and contractures; impairment of sight, nystagmus, and the characteristic disturbance of speech; disproportion between the intense disturbance of motility and the relatively slight disturbance of sensibility; headache, attacks of dizziness, psychical disorders; finally, bulbar symptoms, disturbances of respiration, apoplectiform attacks.

There are only two affections with which the disease can really be confounded, when it is at all pronounced. One of them is *paralysis agitans*, with which it was formerly almost universally confounded, and for which it is even yet occasionally mistaken. Since the publication of Charcot's excellent descriptions, there is no longer any excuse for this mistake. No one who has ever observed closely the two forms of disease will be liable to confound them with one another; the differences are too striking and characteristic. In individual cases, the significance of *particular* symptoms (*e. g.*, the tremor) may, it is true, be somewhat obscure, but the combined clinical picture is, as a rule, so distinctive that the diagnosis is easy.

The following are the most important points for the differential diagnosis. The most essential is the *variety of the tremor.* In paralysis agitans it has the character of short, definite, complicated movements, resembling oscillations; *it appears during perfect rest*, and may also persist during voluntary movement, but is, as a rule, *temporarily controlled by volition;* it *rarely or never extends to the head.* In multiple sclerosis the tremor *ceases entirely during rest; it is excited or aggravated by voluntary movements; the head is invariably involved;* its form is that of longer and more extensive oscillations, or regular to and fro movements, which extend to the entire body when any voluntary exertion is made. Paralysis agitans is a *disease of advanced age;* it may be said to never occur in persons under forty years of age. Multiple sclerosis is a *disease of youth and middle age,* and is seldom or never developed after the fortieth year. In paralysis agitans the paresis is not developed until long after the tremor, perhaps not for a year or more; in multiple sclerosis the paresis or paralysis, as a rule, precedes the tremor, or, at all events, follows it in a relatively short time. In paralysis agitans the cerebral and bulbar symptoms, the scanning speech, the impairment of sight, the nystagmus, the vertigo, the ataxia, the disturbances of sensibility and the paralysis of the sphincters, are wanting, while all of these symptoms belong to the regular and almost invariable phenomena of multiple sclerosis. In addition to these symptoms—which are all-sufficient for most cases—we can also deduce points for the differential diagnosis from other phenomena, which cannot be enumerated in detail here. Above all things, we must bear in mind, that paralysis agitans is a disease which positively is *not* based on gross anatomical changes in the central organ, while in multiple sclerosis, on the other hand, we have to deal with severe anatomical lesions.

F. Schultze has quite recently reported a case of paralysis agitans, in which small sclerotic nodules were found in the posterior columns and the left lateral column of the cervical enlargement, together with much more extensive sclerotic changes in the lumbar enlargement. One might be tempted to conclude from this case, that multiple sclerosis may also be the anatomical basis of the syndrome of paralysis agitans; in my opinion, however, this conclusion would be decidedly too far-fetched. The concurrence in this case was in all probability purely accidental. We can hardly assume the small nodules in the cervical medulla to be the cause of the paralysis agitans, in the face of the numerous and well-authenticated antagonistic facts. Moreover, the much larger nodules in the lumbar enlargement produced no symptoms at all. The only certain conclusion that can be drawn from this case, is that the existence of paralysis agitans does not exclude the possibility of the existence of sclerotic nodules in the brain and the cord. At all events, it is evident from numerous facts that the anatomical basis of paralysis agitans is still entirely unknown, and that in all probability it *is not* to be found in sclerotic nodules in the cord.

The other affection, with which the disease under consideration may possibly be confounded, is sclerosis of the posterior columns, *tabes dorsalis* (ataxie locomotrice). Here, too, the differentiation in pronounced cases is usually very easy, even though ataxia is not at all rare as a symptom of multiple sclerosis.

The points which indicate tabes are the lancinating pains, the girdle-sensation, the disorders of sensation and of the bladder, the reeling when the eyes are closed, the ataxia without marked impairment of the gross strength, the absence of the tendon-reflexes, of the nystagmus, of the impairment of speech, and of the psychical disorders, the very tardy development of pareses, etc. For multiple sclerosis speak: the attacks of dizziness, the cerebral symptoms, the early appearance of pareses and paralyses, the development of contractures, the augmentation of the tendon-reflexes, the tremor (which can easily be distinguished from the ataxia), the nystagmus, the disturbance of speech, the psychical disorders, the apoplectiform attacks, etc. Even in those cases, in which symptoms of tabes accompany the multiple sclerosis, we can, as a rule, recognize the presence of multiple nodules from the paralyses, the contractures, the tremor, the cerebral symptoms, the vertigo, etc.

In consequence of the presence of impairment of speech and nystagmus, Friedreich's cases of hereditary ataxia are, however, more liable to be mistaken for multiple sclerosis, and, in fact, French authors have confounded the two affections together. In this they are unquestionably wrong; in hereditary ataxia we have undoubtedly to deal with a sclerosis of the posterior columns, though, it is true, of a special and not uncomplicated form. It can be distinguished from multiple sclerosis by the fact that the pareses and paralyses, the contractures and the tendon-reflexes, the disturbances of sensibility, the psychical and bulbar symptoms, the vertiginous and apoplectiform attacks, etc., are all wanting.

In the diagnosis of the case reported by Leube (Jenaer Krankenhausbericht) great caution is necessary. The clinical symptoms were those of multiple sclerosis but at the autopsy gray degeneration of the posterior and lateral columns of the cord was found, together with diffuse sclerosis and considerable meningeal changes in the brain. It does not seem to me altogether certain, that the lesion in this case consisted of "the usual changes of a gray degeneration of the posterior columns," but it evidently follows from it that the more or less diffuse affection of the brain must ultimately give rise to the same symptoms as the multiple, insular affection. The same centres are involved in both forms of the disease, and there can be no doubt, that the most essential symptoms of multiple sclerosis are produced by those nodules that are located in the brain.

Some other affections that are also accompanied by active trembling (tremor senilis, mercurialis, saturninus, etc.) may present certain points of resemblance to multiple sclerosis, but the differential diagnosis can always be easily made from the anamnesis alone.

The differentiation from *chorea minor* is also, as a rule, easy, since the volitional tremor differs very essentially from the choreic movements. One case that came under my own observation, has, however, convinced me, that the choreic motor-disturbance can, like ataxia, occasionally occur as a concomitant symptom in multiple sclerosis. In such obscure cases, the true state of affairs will only be recognized, when the existence of multiple sclerosis is demonstrated by other and unequivocal symptoms.

In this connection, too, we must always bear in mind the facts, that anomalous and obscure cases occur, and that, in consequence of the entirely accidental and irregular localization of the nodules in the nervous system, the most peculiar clinical pictures are developed, which may give rise to all sorts of errors in diagnosis.

It has already been stated that the *prognosis* of multiple sclerosis is, under all circumstances, an unfavorable one. Up to the present time no well-authenticated case of recovery has been recorded. The ultimate termination of the affection is consequently a question about which there can be no doubt, although it may not be deemed advisable to inform the patients of the fact. To encourage them stress may be laid on the fact, that marked remissions and ameliorations which persist for a long time, are often observed in the course of the disease. To most of the patients the almost certain prospect of a long duration of the disease, and of the consequent prolongation of life for years, will prove a source of tolerable comfort.

The special prognosis depends of course upon the conditions of each particular case. It is unnecessary to enter into details concerning the manner in which the more or less rapid course of the disease, and the development of particular threatening symptoms, such as cystitis, bed-sores, bulbar symptoms, apoplectiform attacks, etc., can influence the prognosis. This is sufficiently evident from the general principles on which the prognosis of any affection must be based, as well as from the description of the disease given in the preceding pages.

Treatment.

In all essential points, the treatment is the same as that laid down for chronic myelitis. The indications and principles of treatment are the same in both affections, but in multiple sclerosis we must expect the therapeutic effect to be still more limited. In fact, all authors who have written about the treatment of multiple sclerosis agree in the statement, that its results are decidedly discouraging. Charcot, whose experience with this disease has perhaps been more extensive than that of any other man, although his cases, it is true, were mostly advanced and hopeless, has almost nothing but failures to report. Arsenic, belladonna, bromide of potassium, ergot, and strychnine, proved entirely useless in his hands; chloride of gold and phosphate of zinc aggravated rather than relieved; nitrate of silver, on the contrary, had a distinctly favorable effect, which, however, was only transitory; hydropathy proved decidedly useful in one case. He recommends that further investigations be made with electricity. Hammond advises chloride of barium in doses of 0.05 grm. (three quarters of a grain) three times a day. Schuele saw a transitory improvement after the cold-water treatment, and I have myself had a similar experience. On the other hand, several cases have been recorded (Otto, Baerwinkel, Berlin), which were aggravated by the use of the thermæ. In one case that was treated in the hospital in this city, marked improvement set in under the use of subcutaneous injections of arsenic; at a later period this case was distinctly benefited by the galvanic current. As has already been stated, no authenticated case of recovery is known.

These unsatisfactory results, however, must not lead us to abandon all therapeutic efforts, especially in more recent cases; in these a favorable result may occasionally be obtained, just as in simple chronic myelitis. In the first place, the treatment recommended for chronic myelitis must be steadily and patiently followed out; the persistent employment of the galvanic current, of hydropathy, and of nitrate of silver, is especially to be commended. If these remedies prove useless, there will always be time and opportunity for extended therapeutic trials of all possible measures, and perhaps accident will lead to the discovery of an efficient remedy for this, at present hopeless, disease.

12. *Gray Degeneration of the Posterior Columns—Sclerosis of the Posterior Columns—Tabes Dorsalis — Ataxie Locomotrice Progressive* (Duchenne)—*Leukomyelitis Posterior Chronica.*

Ollivier, l. c. 3. éd. Tom. II. p. 454. Obs. 132 and 133.—*Cruveilhier*, Anat. patholog. Tom. II.—*Romberg*, Lehrbuch der Nervenkrankheiten. I. 3. Abth. S. 184. 2. Aufl. 1851.—*Wunderlich*, Handbuch d. Pathol. u. Therapie. 2. Aufl. 1854. *Axenfeld*, In the Dict. encyclop. des Sciences médic. T. VII. p. 56. 1867.—*Trousseau*, Medic. Klin. des Hôtel-Dieu, Deutsch von Culmann. II. p. 544. 1868.—*Benedikt*, Elektrotherapie. S. 331. 1868.—*Hasse*, l. c. 2. Aufl. S. 713. —*Hammond*, l. c. 3d edit. p. 360.—*Leyden*, Klinik d. Rückenmarkskrankheiten. II. 2. Abth. S. 324. 1876.

W. *Horn*, De tabe dorsuali prælusio. Berol. 1827.—*Decker*, De tabe dorsuali. Diss. Berolin, 1838.—*Brach*, Med. Zeitung des Vereins f. Heilk. in Preussen. 1840. Nr. 45, und 1842. Nr. 3, 4.—*Jacoby*, Exempl. tabis dorsual. epicrisi ornatum. Berolin, 1842.—*Kuschel*, De tab. dors. Berol. 1844.—*Steinthal*, Beitr. z. Geschichte u. Pathol. d. Tab. dors. Hufeland's Journ. Band 98. 1844.—*Todd*, Cyclop. of Anat. and Physiol. III. 1847.—*Tuerck*, Ueber primäre Degeneration einzelner Rückenmarksstränge. Sitzungsber. d. k. Akademie zu Wien. Mathem. naturw. Classe. XXI. Jahrg. 1856. Heft I. u. II. S. 112.—*Duchenne (de Boulogne)*, De l'ataxie locomotr. progressive. Arch. génér. de Méd. 1858. Déc. 1859, Janv., Avril.—Electrisat. localisée. 2. éd. 1861. 3. éd. 1872.—*Harless*, Physiol. Vermittlungswege paralytischer und paret. Erscheinungen. Bayr. ärztl. Intelligenzbl. 1858. Nr. 13.—*Oppolzer*, Krankh. des R.-M. Spitalszeit. 1859. Nr. 21.—*Trousseau*, De l'atax. locomotr. progress. Union méd. 1861. Nr. 12, 14, 20.—*Bourdon*, Cas d'atax. loc. Gaz. hebdom. 1861. Nr. 41.—*Bourdon* et

Luys, Études clin. et histol. etc. Arch. génér. Nov. 1861. Avril, 1862.—*Teissier*, De l'ataxie musculaire. Gaz. méd. de Lyon. 1861, Dec. 1862, Janv.—*Dumenil*, Union méd. 1862. Nr. 17.—*Oulmont* et *Luys*, Union méd. 1862. Nr. 41. —*Charcot* et *Vulpian*, Atrophie des cordons postér. etc. Gaz. hebdom. 1862. Nr. 16, 18.—*Marotte* et *Luys*, Union méd. 1862. Nr. 67.—*Trousseau* et *Sapey*, ibid. Nr. 88, 89.—*Duguet*, Atax. locom. de forme hémiplég. Ibid. Nr. 122.—*Marius Carré*, De l'atax. locom. Thèse. Paris, 1862.—*M. Carré*, Gaz. méd. de Lyon 1864. Nr. 15, 20; Nouv. recherches sur l'atax. loc. progr. Paris, 1865. —*Dujardin-Beaumetz*, De l'atax. loc. Paris, 1862.—*Eisenmann*, Die Bewegungsataxie. Wien, 1863.—*Friedreich*, Ueber degenerative Atrophie der spinalen Hinterstränge. Virch. Arch. Bd. 26 u. 27. 1863.—*Leyden*, Die graue Degeneration der Hinterstr. de R.-M. Berlin, 1863. Deutsche Klinik. 1863. Nr. 13. Virch. Arch. Bd. 40. 1867.—Ueber Muskelsinn und Ataxie. Ibid. Bd. 47. 1869.—*Westphal*, Tabes dorsual. u. Paralysis universal. progress. Zeitschr. f. Psych. XX. 1863. u. XXI. S. 361. 1864.—*Westphal*, Ueber Erkrankung des R.-M. bei der allg. progress. Paralyse der Irren. Virch. Arch. Bd. 39 u. 40. 1867.—*Charcot* et *Vulpian*, Deux cas de Sclérose des cord. postér. etc. Gaz. méd. 1863. Nr. 14.—*Axenfeld*, Des lésions atroph. de la moëlle épin. Arch. génér. 1863, Aug. Oct.—*R. Remak*, Ueb. Tabes dorsalis. Deutsche Klin. 1862. Nr. 49. Berl. klin. Wochenschr. 1864. Nr. 30, 41.—*Benedikt*, Wien. med. Wochenschr. 1862. No. 44–48. 1864. Nr. 23, 30, 37.—*Teissier*, De l'at. loc. Gaz. méd. de Lyon. 1864. Nr. 19.—*Cornil*, Gaz. méd. de Par. 1864. Nr. 19.—*Duchenne*, Rech. clin. sur l'état pathol. d. grand Sympath. etc. Gaz. hebd. 1864. Nr. 8, 10.—Diagnost. differ. des affect. cérébell. et de l'atax. loc. Gaz. hebd. 1864. Nr. 29, 31.—*Cel. Bernard*, De l'at. loc. Thèse. Strasbourg, 1864. —*Finkelnburg*, Beobb. üb. d. paralyt. Bewegungsataxie. Berl. klin. Wochenschr. 1864. Nr. 53.—*Topinard*, De l'at. locom. Paris, 1864.—*Jaccoud*, Les paraplégies et l'atax. du mouvement. Paris, 1864.—*Fr. Vaneschi*, De tabe dors. Diss. Berol. 1864.—*Remak*, Allgem. med. Centralz. 1862, Dec. 1863, Dec. 1864. Nr. 83.—*Spaeth*, Zur Lehre von d. Tab. dors. Dis. Tübingen, 1864.—*Nachtweyh*, *Kauert*, *Mette*, De tabe dors. Diss. Berol. 1864.—*Fr. Boening*, Beobb. über progress. Bewegungsataxie. Deutsche Klinik. 1865. Nr. 1, 5, 8.—*Bouchard*, Des lésions anatom. de l'at. loc. progr. Lyon, 1865.—*Nothnagel*, Berl. klin. Wochenschr. 1865. Nr. 17.—*O. Frohwein*, Diss. Erlangen, 1865.—*Oppolzer*, Wien. med. Wochenschr. 1866. Nr. 26–28.—*Charcot* et *Bouchard*, Douleurs fulgur. de l'ataxie sans incoordin. des mouv., Sclérose commençante des cord. post. Gaz. méd. 1866. Nr. 7. Compt. rend. de la Sociét. de Biolog. 1866.—*Fabre*, Physiol. pathol. et diagn. de l'atax. etc. Gaz. des hôp. 1866. Nr. 107, 108.—*Althaus*, On Epilepsy, Hysteria, and Ataxy. London, 1866.—*Lockhart Clarke*, On Locom. Ataxy. St. George's Hosp. Rep. I. 1866. Brit. Med. Journ. 1869. July 3, 31; Sept. 25; Dec. 11.—*H. Fabricius*, Diss. Berlin, 1867. *E. Cyon*, Zur Lehre von der Tabes dors. Berlin, 1867. Virch. Arch. Bd. 41. 1867.—*Frommann*, Unters. üb. d. normale u. pathol. Anat. des R.-M. II. Jena, 1867.—*Robitzsch*, Zwei ungewöhnl. Fälle von Tab. d. Diss. Berlin, 1867.—E.

Schulze, Ueb. d. Aetiologie der Tab. dors. Diss. Berlin, 1867.—*Vulpian*, Etat des nerfs sensit., des gangl. spinaux, etc. Arch. d. Physiol. norm. et pathol. I. p. 128. 1868.—*Vulpian*, Retard des sensat. dans les cas de sclérose, etc. Ibid. I. p. 463. 1868.—*Larroche*, Thèse. Montpellier, 1868.—*Dubois*, Étude sur quelques points de l'at. loc. Thèse. Paris, 1868.—*Axel Jaederholm*, Studien üb. d. graue Degener. des R.-M. Nord. medic. Arkiv. I. Nr. 2. 1869.—*Th. Laycock*, Influence of Libid. Excess on the Causat. of Locom. Ataxy. Dubl. Quart. Journ. May, 1869.—*Bracht*, Zur Symptomatol. d. Tab. d. Diss. Berlin, 1869.—*Winsor*, Case of Loc. At. Bost. Med. and Surg. Journ. 1870, Oct. 6.—*Meredith Clymer*, Some Points in the Clinical History, etc. New York Med. Record. 1870, Febr. 1.—*Arndt*, Mittheil. üb. die Histol. der grauen Degen. u. s. w. Berl. klin. Woch. 1870. Nr. 11.—*Pierret*, Altérat. de la subst. grise dans l'at. locom. progr. Arch. de Physiol. III. p. 599. 1870.—*Pierret*, Sclérose des cord. postér. dans l'ataxie. Ibid. IV. p. 364. 1871, 1872.—Cas de Sclér. primit. du faisceau médian des cord. postér. Ibid. V. p. 74. 1873.—*Sam. Wilks*, Ataxia. Guy's Hosp. Rep. III. Ser. Vol. XVII. p. 198. 1872.—*Tigges*, Ueb. mit Tab. dors. complic. Psychose. Allg. Zeitschr. f. Psych. Bd. 28. S. 245. 1872.—*v. Krafft-Ebing*, Ueb. Tab. dors. mit finaler Geistesstörung. Ibid. S. 578.—*H. Leonhardt*, Ueb. Tab. dors. Diss. Berlin, 1872.—*Vossius*, Beitr. z. Symptomat. d. Tabes. Diss. Berlin, 1873.—*Charcot*, Leçons sur les malad. du syst. nerveux. II. Sér. 1. fasc. 1873.—*C. Lange*, Nord. medic. Ark. IV. 1872. S. Virchow-Hirsch, Jahresber. pro 1872. II. S. 79.—*Martin*, De l'atax. loc. pr. Thèse. Paris, 1874.—*Voisin*, Progrès méd. 1875. Nr. 10.—*Hayem*, Atax. loc. progr. Nécropsie. Gaz. méd. 1876. Nr. 19.—*Erdmann*, Z. Beurtheilung des Verlaufs tabet. Lähmungen. Jahresber. d. Gesellsch. f. Natur- u. Heilk. in Dresden. Oct. 75. Juni, 76. S. 56. 1876.—*Friedreich*, Ueb. Ataxie mit besonderer Berücksichtigung der hereditären Formen. Virch. Arch. Bd. 68. 1876. Bd. 70. 1877.—*Kellogg*, Two Cases of Loc. At. in Children. Arch. of Electrol. and Neurolog. Vol. II. p. 182. 1875.—*Du Castel*, Sclérose primit. des cordons de Goll. Gaz. méd. 1874. Nr. 3.—*Fournier*, L'ataxie locomotrice d'origine syphilitique. Gaz. méd. 1876. Nr. 53.

Landois u. *Mosler*, Neuropathol. Studien. Berl. klin. Wochenschr. 1868. Nr. 41. —*E. Remak*, jun., Ueb. zeitliche Incongruenz der Berührungs-und Schmerzempfindung bei Tab. dors. Arch. f. Psych. u. Nervenkrankh. IV. S. 763. 1874. —*Naunyn*, Ueb. eine eigenthüml. Anomal. d. Schmerzempfindung. Ibid. S. 760.—*Osthoff*, Verlangsamung d. Schmerzempfind. bei Tabes. Diss. Erlangen, 1874.—*Hertzberg*, Sensibilitätsstörung bei Tabes dors. Diss. Jena, 1875.

Galezowsky, Des troubles oculaires dans l'atax. loc. progr. Gaz. des hôp. 1874. Nr. 85.—*Leber*, Ueber graue Degener. d. Sehnerven. Arch. f. Ophthalm. XIV. 2. S. 177. 1868.—*Kiesselbach*, Zur grauen Degen. der Sehnerven bei Erkrank. des Cerebrospinalsyst. Diss. Erlangen, 1875.—*Hempel*, Ueb. Spinalmyosis Arch. f. Ophthalm. XXII. 1. 1876.—*Foerster*, Im Handb. d. gesammt. Augenheilk. von *A. Graefe* u. *Saemisch*. VII. 1. S. 131. 1876.—*A. Pierret*, Essai sur les symptomes céphaliques du Tabes dorsalis. Paris, 1876.

Delamarre, Troubles gastriques dans l'at. l. pr. Thése. Paris, 1866.—*Petitjean*, Crises gastriques dans l'ataxie. Thèse, 1874.—*Grainger Stewart*, Peculiar Form of Gastric Crisis in Loc. Ataxy. Medic. Tim. 1876. Oct. 7.—*M. Raynaud*, Crises néphrétiques dans l'at. loc. Arch. génér. Oct. 1876.—*Féréol*, Sur quelques sympt. visceraux-laryngo-bronchiques de l'atax. Gaz. hebdom. 1869. Nr. 7.—*Jean*, Troubles atax. du coté du larynx et du phar. Gaz. hebdom. 1876. Nr. 27.—*Chvostek*, Tabes complic. mit Atrophie des linken Halssympath. u. s. w. Allgem. Wien. med Zeit. 1874.—*M. Cuffer*, Hémiatrophie d. l. langue surven. d. l. cours d'une ataxie. Union méd. 1875. Nr. 72.

Charcot, Sur quelques arthropathies, qui paraissent dépendre, etc. Arch. d. Physiol. I. p. 161. 1868; u. II. p. 121. 1869.—*Charcot* et *Joffroy*, Lésion de l. subst. grise dans un cas d'arthropath. ataxique. Ibid. III. p. 306. 1870.—*Ball*, Arthropathies, etc. Gaz. des. hôp. 1868. Nr. 128 u. 1869. Nr. 58–62.—*Hervey*, Arthrop. liée à l'at. Ibid. 1868. Nr. 33.—*Blum*, Des arthropathies d'origine nerveuse. Thèse. Paris, 1875. pp. 19–50.—*Buzzard*, Locomot. ataxy with anomalous joint affections. Lancet, 1874. Aug. 22.—*Charcot*, Luxat. pathol. et fractures spontanées multiples dans l'atax. Arch. d. Physiol. VI. p. 166. 1874.—*Forestier*, Arthropathies, fractures et luxat. consécut. Paris, 1874.—*Brochin*, Des lésions osseuses chez les atax. Gaz. des hôp. 1875. Nr. 12.—*Raymond*, Note sur les arthrop. de l'at. Gaz. méd. 1876. Nr. 8.

R. Remak, Ueb. d. Behandlung der Tab. d. mit d. const. galv. Strom Med. Centralz. 1858. Nr. 29. 1862. Dec. 3.—*Wunderlich*, Behandlung der Spinalparalysen mit Silbersalpeter. Arch. d. Heilk. 1861 u. 1863.—*Charcot* et *Vulpian*, Emploi du nitrat d'arg. etc. Bullet. d. Thérap. Juin, 1862.—*Eulenburg*, Erfolg v. Arg. nitr. Verh. d. Berl. med. Ges. 1866. Heft. 2. S. 142.—*Hitzig*, Schädliche Wirkung des Arg. nitr. bei Tabes dors. Berl. klin. Woch. 1867. Nr. 31. *Dujardin-Beaumetz*, Emploi du phosphore, etc. Bull. génér. Thérap. 1868.—*Siredey*, Action du bromure de potass. dans l'atax. l. pr. Ibid. 1872. Août 30.—*Delmus*, Six observat. d'atax. loc. Journ. de Méd. d. Bord. Mars, 1865. (cold-water treatment).—*v. Krafft-Ebing*, Ueb. Heilung und Heilbarkeit der Tabes durch den galv. Strom. Deutsch. Arch. f. klin. Medic. IX. 1872.—*Karmin*, Fall von geheilter Tabes. Wien. med. Woch. 1868. Nr. 35.—*Onimus*, De l'emploi des courants contin. dans le trait. de l'at. Gaz. des hôp. 1868. Nr. 116–119.—*Delmar*, Trait. de l'at. loc. Rev. méd. 1874. Nr. 38. 39.—*Waldmann*, Behandlung d. Tabeskranken als Anhalt für Aerzte u. Kranke. Halle, 1872.—*Mendel*, Beh. der Tabes dors. Deutsch. Zeitschr. f. prakt. Medic. 1874. Nr. 39.—*Fr. Richter*, Ibid. 1874. Nr. 48.—Ueb. Temperatur u. Mechanik der Badeformen bei Tabes, etc. Ibid. 1875.—*Caster*, Oxide of Silver in Locom. Ataxy. Philad. Med. and Surg. Rep. 1875. Dec.

Refer, furthermore, to the text-books on Electrotherapeutics, Balneotherapeutics, and Hydrotherapeutics, also to the larger hand-books and encyclopædias of Special Pathology and Therapeutics, all of which contain more or less detailed accounts of the pathology and therapeutics of Tabes.

History.

We begin the discussion of those affections of the spinal cord which are limited to certain definite portions of the transverse section of the cord, but which reach a very varied longitudinal extent, with the consideration of that form thereof which is best known and most studied, viz., gray degeneration or degenerative atrophy (Friedreich) of the white posterior columns. This is the same affection which is now almost universally designated as "tabes dorsalis," in Germany, the term which was formerly used for atrophy of the spinal cord in general being thus restricted to this special form.[1]

This disease was undoubtedly known to the most ancient physicians, and frequently enough came under their observation. Among the forms of spinal disease which are more or less definitely spoken of, under the name of tabes, or of phthisis ischiadica, by Hippocrates, Galen, Bonetus, and others, there were undoubtedly also cases of sclerosis of the posterior columns. But it is hardly worth the while to trace out the reports of the older physicians on these forms of disease, for we shall nowhere find anything like an accurate characterization of the same, or a correct distinction between the different forms.

Neither would it prove any more profitable to follow up all the descriptions of tabes dorsalis, phthisis of the spinal cord, etc., which at the end of the last and the beginning of the present century were ascribed to the influence of sexual excesses and spermatorrhœa, and which reached their climax in the extravagancies of Lallemand.

It was not until the third and fourth decade of the present century that occasional more exact observations appeared, which are to be regarded as the first foundation stones of the doctrine of tabes dorsalis. The anatomical observations of Hutin (1827) and Monod (1832) undoubtedly belong to this disease, although the latter were still cited by Ollivier as cases of hypertrophy of the gray substance. Cruveilhier, in his celebrated atlas, besides admirable anatomical representations, also gives a series of histories of the disease.

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[1] More generally known in English as progressive locomotor ataxy.—TRANS.

But it was principally German physicians who, in the next two decades, occupied themselves in working up the subject of tabes, presenting a more precise picture of the disease, and collecting the results of post-mortem examinations belonging thereto. The Dissertation of W. Horn (1827) may be regarded as the starting-point of these labors. In the subsequent classical description given by Romberg all the principal symptoms of the disease are enumerated, and he even draws the distinction between the peculiar form of motor disturbance that is present and true paralysis. The anatomical changes were more accurately made known through the investigations of Froriep, E. Horn, Jacoby, and others, and the work of Steinthal (1844) gave a tolerably complete representation of the pathology of tabes, even though it does include under that head a good deal that is foreign thereto. German physiologists (Joh. Mueller, Spiess) recognized the peculiar form of motor disturbance which Bouillaud (1845) and Todd (1847) correctly designated as a disturbance of co-ordination, and not as paralysis. The description of the disease given by Wunderlich (1854) leaves but little more to be desired. Rokitansky and Tuerck advanced our knowledge of the anatomical alterations by their microscopic examinations. In Germany, therefore, as early as the beginning of the latter half of this century, the history and pathological anatomy of tabes dorsalis was firmly established in its main features, and was sufficiently well known to the medical public.

It was therefore a somewhat peculiar and certainly an unjustifiable undertaking on the part of Duchenne, in the year 1858, to describe the disease as an entirely new one and under a new name (Ataxie locomotrice progressive). Entirely ignoring all the labors of his predecessors, he seeks the seat of the disease, on theoretical grounds, in the cerebellum, whereas it had long since been found to lie in the spinal cord.

At the same time, we must certainly admit that Duchenne gave a most admirable description of the disease, one more accurate than had previously been furnished; that he characterized and emphasized the first stage of the disease, though, perhaps, in a somewhat one-sided manner; that he was the first more accurately to establish the idea of a disturbance of co-ordination (ataxy), and to demonstrate unequivocally, by means of the dynamometer, the preservation of the gross muscular strength.

Duchenne's work unquestionably excited an extraordinary degree of interest in all directions, and gave occasion to such numerous works on Tabes dorsalis that the literature of the subject has already swelled to dimensions which we can hardly compass.

Numerous investigations and discussions first arose in France over the "new" disease, which, under the powerful protection of Trousseau, soon claimed general recognition. An extensive amount of material was accumulated, partly in the form of comprehensive representations of the disease (Dujardin-Beaumetz, Mar. Carré, and others), partly in the form of reports of cases (Bourdon, Luys, Oulmont, Teissier, Dumenil, Charcot, Vulpian, and others), thus clearing up the views held on the subject. Stimulated by a prize question proposed by the Academy, several larger works on Ataxy appeared in 1864 and 1865, which treated the question in an exhaustive and in part in a most admirable manner (Topinard, Mar. Carré, Jaccoud).

But in Germany also numerous and admirable works appeared. In the year 1863 three very noteworthy works, of great value in the history of tabes, were put forth by Friedreich, Eisenmann and Leyden, to which were afterwards added many larger and smaller communications and investigations (Westphal, Frommann, Spaeth, Remak, Benedict, Finkelnburg, Boening, and others).

Since then the production of works on tabes has hardly, as yet, suffered any diminution; particularly in France and Germany, each year brings a series of valuable contributions, while, aside from the admirable labors of Lockhart-Clarke, English literature actually contains but very little on this subject.

The last few years have materially advanced and deepened our knowledge of tabes, although we are doubtless still far from having arrived at a final conclusion of the same. The anatomical changes have been more accurately investigated in their finer histological details, and in their more precise localization in a transverse section of the cord; the picture of the disease has been more sharply defined and relieved of unimportant and accidental features; the careful observation of details has taught us to recognize a series of rarer but not less interesting symptoms; the theory of the disease and of its individual symptoms has been elucidated by thorough and lively discussions, although it has not yet been finally established; the diagnosis has been rendered easier and more delicate, the distinction between this disease and neighboring and kindred affections has been materially advanced; and the prognosis, too, which was formerly so gloomy, has been essentially modified by noteworthy advances in the therapeutics of the disease. The evidences of this are to be found in the writings of the past ten years, as cited in the above table of literature.

It would carry us too far if we were to follow, even quite cursorily, the historical development of the various scientific victories gained in connection with this subject of tabes; if we were to attempt to show how the anatomical views gradually developed themselves up to the present standpoint; how the theoretical views of the entire disease and of single symptoms thereof (for example, the ataxy) slowly assumed form, amidst the most lively disputations; what stages of development the therapeutics of the disease itself has undergone, etc. The reader who desires information on this subject must make his own special studies thereon.

We desire only to be permitted a few remarks on the most appropriate *designation* for the disease. None of the designations thus far in use can be regarded as entirely appropriate or exhaustive. The term "tabes dorsalis" is hardly to be justified by the history of the disease and not at all so by its anatomy, although it at least has the advantage of antiquity. The term "progressive locomotor ataxy" is still more unfortunately chosen, as it is derived from one single symptom which often does not appear until the disease has existed for years, and is sometimes also present in other diseases. From an anatomical point of view, to which we should endeavor, as far as possible, to adhere, the term "gray degeneration or degenerative atrophy of the posterior columns of the cord" might the most readily be accepted.

Aside from its length and awkwardness, however, it is open to the objection that it only designates a comparatively late stage of the anatomical alterations, and that, in all probability, it is not even exhaustive, inasmuch as an extension of the process to the gray substance and to neighboring portions of the lateral columns probably constitutes the rule. The same objection applies to the name "Leukomyelitis posterior chronica," although this in a happy manner emphasizes the chronic inflammatory character of the process.

Still, in the end, it is all the same what we call a thing, the main point being to know and to determine what we wish to have understood under a certain designation. If we can unite on the clinical and anatomical characterization of the disease—and I think that at the present day there is no serious difficulty about this with regard to the disease under consideration—we shall also be able to come to an understanding as to an appropriate designation for the same. In my opinion—until something better is found—it might be most appropriate to adhere to the old name of tabes dorsalis, which was naturalized by Romberg and has also of late been extensively used by the French, it being distinctly understood that the term is to be limited to the designation of that form of disease described in the following pages. "Sclerosis of the posterior columns" ("Hinterstrangsklerose") is another not very prejudicative designation which might recommend itself, on account of its brevity, to those who favor a name selected from an anatomical standpoint.

Definition.

Under the name of tabes dorsalis we understand a disease of the spinal cord which runs a slow course, which arises principally during youth and middle age, and which in all probability belongs to the group of chronic myelitis.

It is anatomically characterized by ribbon-like sclerosis of the white posterior columns, leading to gray degeneration, and probably also by later participation on the part of the adjoining portions of the white lateral columns and the gray posterior horns; still, nothing more accurate is established with regard to the more or less regular and uniform participation of these parts in the process. The affection generally begins in the lumbar region, and may extend throughout the entire cord as far as the upper cervical portion, and even into the medulla oblongata.

Clinically the disease is characterized by a first stage, which often stretches over many years, and which is marked by lancinating pains, disturbances of certain cerebral nerves (the optic and the nerves supplying the muscles of the eye), paræsthesias in the legs, on the trunk, and in the domain of the ulnar nerve, debility, a tendency to weariness and unsteadiness in the legs, weakness of the bladder and of the generative organs.

The second stage is that of the fully-developed disease, in which, aside from a more or less high degree of disturbance of sensation (pain, anæsthesia, paræsthesia, sensation of a tight girdle, etc.), and aside from weakness of the bladder and the genitals and the disturbance of certain cerebral nerves, a distinct and characteristic disturbance in the co-ordination of motion becomes prominent, without the gross power of movement being, to any considerable degree, diminished (ataxy). The cerebral functions still remain intact.

Finally, there is the third stage, that of progressive spinal paralysis, characterized by actual paralysis, muscular atrophy, contractures, troubles of the bladder, bed-sores, and, at last, general marasmus.

It is a disease of a decidedly progressive character, although it is not exactly rare for it to come to a standstill and improve, and in some few instances actual recovery even takes place.

It is always a question of a disease of long duration, which is to be counted by years, and not rarely by decades.

Etiology and Pathogenesis.

Tabes is one of the most frequent affections of the spinal cord. The *predisposition* to the same must, therefore, be tolerably wide-spread.

In developing this predisposition, the *neuropathic tendency* of numerous individuals undoubtedly plays a very prominent part. The increasing frequency of nervous diseases generally, and of tabes in particular, the large number of nervous and hysterical women, result in the birth of an ever-increasing number of individuals with a tendency to nervous diseases. In the family history of those affected with tabes it will not be rare to be able to demonstrate the occurrence of hysteria, epilepsy, mental diseases, migraine, or it may be only of violent fits of anger, drunkenness, or anomalies in the form of the skull, malformations of the external ear, etc. Such instances have been narrated by Trousseau, Rosenthal, Topinard, and others. The condition of things most rarely found is that of the direct hereditary transmission of the disease ; the most striking instance of this is given by Carré, who saw eighteen cases of the disease occur in one family, in three generations. As a rule, parents transmit to their children only the predisposition to tabes. This was also especially true in the remarkable observations communicated by Friedreich. In three different families several brothers and sisters were each time attacked with the disease at almost the same age, without the parents themselves having suffered from it. The only thing we can suppose is that in such cases a certain weakness and irritability of the nervous system, perhaps also a species of impediment to the development of certain portions of the same, is hereditarily transmitted, which renders the nervous system less capable of resisting external injuries.

At the same time, according to my experience, there is a very large number of cases of tabes in which there is decidedly no hereditary neuropathic tendency, the patients coming from families that were, nervously, perfectly well.

Almost all observers concur in the statement that the *male sex* shows a decidedly greater predisposition to tabes than the female. I can fully confirm this from my own observation; among eighty-three cases of undoubted tabes seen by me, seventy-four were men and only nine women. It is probable that this greater predisposition is largely owing to the fact that men are far more exposed than women to the strongest direct causes of tabes—taking cold and sexual excesses. Still, this might not entirely account for so striking a preponderance of males.

The statistics of different authors, although without exception confirming the fact of a marked preponderance of the disease in men, still show great differences in the proportionate frequency of the disease in the two sexes. I here place together some of these statements:

	Men.	Women.				
M. Carré	42	18	— about	in the	proportion of	2⅓ : 1.
Topinard	21	4	"	"	"	5 : 1.
Steinthal	42	6	"	"	"	7 : 1.
Eisenmann	46	20	"	"	"	2⅓ : 1.
Schulze	37	9	"	"	"	4 : 1.
Cyon	149	43	"	"	"	3½ : 1.
Leonhardt	12	11	"	"	"	1 : 1.
Erb	74	9	"	"	"	8 : 1.

It is not permissible to add together these numbers, as they refer, in part, to the same cases. Of course these depend very much on the accidental supply of material, and it is only thus that the striking statement of Leonhardt can be explained, which, it is true, only relates to quite small numbers. At all events, the preponderance of males is established beyond all doubt.

Tabes is a disease of *youth and middle age;* by far the majority of individuals are at the top of the hill of life when the disease begins—between their thirtieth and fiftieth year. In the third decade of life, too, cases of the disease are tolerably common; before the twentieth and after the fiftieth year, however, they are very rare. This, too, might have some connection with the above-named causal conditions.

Among sixty-eight observations of my own, in which the *beginning* of the disease could be determined with some certainty, it was found to occur as follows:

Between the ages of	11	and	20,	three	times.
" "	21	"	30,	thirteen	"
" "	31	"	40,	thirty-one	"
" "	41	"	50,	eighteen	"
" "	51	"	60,	three	"

The statistics collected by other observers (Topinard, Carré, Cyon, etc.) correspond pretty accurately with these.

No doubt it is owing to the greater influence of external injurious causes that *certain occupations* furnish a larger proportion than others to the number of those attacked with tabes. This is said to be the case especially in those persons who, by reason of their calling, are obliged frequently to expose themselves to all extremes of the weather, cold and wet—and who are subjected to all sorts of bodily hardships, such, for instance, as commercial travellers, hunters, engineers, soldiers, firemen, workers on ice, railroad conductors, fishermen, etc. At the same time, all these are more subordinate and accidental causes.

Bodily and mental overexertion are certainly to be counted among the injuries which may occasion an increased predisposition to tabes. The increased demands of the fight for existence, the excitements and exertions of modern social life, are certainly in no small degree responsible for the more frequent occurrence of tabes at the present day. This explains the greater frequency of the affection in large cities and in the higher walks of life, in which mental overexertion, want, cares, anxiety, failures in life, and the like, often play a very prominent rôle. All these things are to be regarded as weakening to the body, and especially to the nervous system, and thereby as predisposing to disease.

The same predisposing influence may be exerted (E. Schulze) by previous *acute and chronic diseases* (typhus, intermittent fever, and the like), whether occurring but once or repeatedly. A similar effect may also be claimed for syphilis; for the existence of an actual, specific, syphilitic sclerosis of the posterior columns may yet well be doubted. But in view of the comparative frequency of both forms of disease, it is hard to exclude an accidental concurrence of the same.

Sexual excesses and *onanism* are certainly of no slight significance, at least in the development of a predisposition to tabes. We have already previously expressed ourselves in detail on this point (page 147), and what was said there holds especially good with regard to tabes, the most frequent of all chronic diseases of the spinal cord. We do not entertain the slightest doubt that such sexual excesses, of the most varied kind, over-irritate and weaken the spinal cord, and at all events make it more susceptible to the influence of direct injuries.

Among the *direct causes* of tabes the first that may be mentioned is *taking cold.* It is regarded by some (Leyden, Leonhardt) as the most frequent, in fact as the almost exclusive cause of tabes; but while fully recognizing the undeniable potency of this injury, we must yet look upon this view as being extreme.

It is notorious that individuals who are otherwise quite well and not burdened with any neuropathic tendency, vigorous people from among the working classes, those working in water and ice, foresters, soldiers, etc., frequently notice the first symptoms of tabes after they have been, once or repeatedly, exposed to some serious injury in the way of taking cold. Instances are not rare in literature going to prove that the disease has come to an outbreak as the result of inundations, after a person's falling into the water or getting wet through, after a wet, cold bivouac, after occupying damp living-rooms or workshops, and the like.

This holds particularly true for persons of a nervous temperament, or who are exhausted and reduced through exposure, sexual excesses, mental disturbances, night-watching, and the like; in such persons even the slighter causes for taking cold may be effective, such as sleeping in a damp room, etc.

We possess no more plausible explanation of the *modus operandi* of "taking cold" in the production of tabes than in the numerous other forms of disease which may likewise be brought about by the same cause. It is true that various attempts at an explanation of the same have been set up, which are very well meant, in themselves, but do not stand the test of severe criticism. We consider it unnecessary to enter into any more detailed consideration of this question, and content ourselves with merely confirming the fact that, in many cases, "taking cold" can be shown to be the immediate exciting cause of tabes.

Bodily overexertion and the exhaustion induced thereby, *hardships* of all sorts, are often stated as being the direct occasion of tabes. Their efficacy appears to be especially manifest if, at the same time, an opportunity is given for taking cold, and active mental disturbances likewise exist. I have twice seen tabes break out in consequence of fatiguing and exciting business journeys undertaken during severe cold winter weather. The fact that severe marches and other hardships, combined with mental activity and excitement, very readily lead to tabes, has been most unequivocally proved by all the campaigns of this century, after which the development of tabes has always been observed to be especially frequent. To be sure, it is hard to determine in such cases the share of responsibility to be assigned to each of the injurious conditions. At the same time, I believe that the mere overexertion of itself, especially in persons disposed that way, may prove effective to this end. (For an example of the same see Waldmann, p. 74.)

All this holds good, to a still higher degree, with regard to *sexual over-excitement and excesses*, which may also sometimes become the direct causes of tabes. Formerly these were regarded as the principal and almost the only causes of tabes, and every poor victim of the disease lay under the suspicion of having led a dissolute life. This was certainly wrong; it is but a limited number of cases that are unquestionably caused in this way. But it is at least equally wrong to deny this etiological connection entirely, or in great part. Aside from the predisposing influences, which doubtless constitute the main feature, cases are also not rare in which the first manifestations of tabes immediately followed great sexual excesses (for instance, in newly-married persons). This view is even somewhat favored by the mere fact of the far more frequent occurrence of tabes in men, and during the period of their greatest sexual activity.

Onanism, carried to excess, acts in the same way as the excessive natural gratification of the sexual appetite; the same is true of very frequent pollutions, pollutiones diurnæ, spermatorrhœa, and the like. And yet, with regard to just these last processes, it is often hard to determine what is the cause and what may perhaps already be the effect of the disease beginning to be developed.

With regard to the method of operation of sexual excesses it is also difficult to arrive at any plausible theory which shall have more weight than that of a mere conjecture. We shall touch briefly, below, upon the opinions expressed on this subject.

Extreme and lively *emotional activity* appears now and then capable of being the starting-point of tabes; at least this is asserted to be true of lively fright, terror, continued anxiety, cares, repeated anger, etc. It is well known that these emotions are often accompanied with serious disturbances of innervation (trembling and weakness of the limbs, vaso-motor disturbances, etc.). It is at least not inconceivable that an increase and repetition of these disturbances might also finally lead to actual disease of the spinal cord. But nothing accurate can be said on this point.

It is hard to determine what relation certain *acute diseases* bear to the tabes which follows them, whether they are merely predisposing, or whether they directly light up the disturbances of nutrition in the spinal cord. The fact is, however, that evidences of tabes are often developed, sometimes more quickly and sometimes less so, in the train of typhus, articular rheumatism, acute pneumonia, etc. The same thing holds true of repeated labors and abortions, of serious losses of blood, too long continued lactation, etc.

Ataxy has also been repeatedly observed among the nervous disturbances which so often appear in the train of *diphtheria*; but it may appear doubtful whether we here have an actual affection of the posterior columns of the cord, or perhaps another form of ataxy; still, the observation reported by Jaccoud (l. c., p. 631) appears to me to speak positively for the first supposition. I am myself at present observing an interesting case, in a child nine years of age, which, after diphtheria of the fauces, at first showed paralysis of the pendent palate, paresis of accommodation, and insufficiency of the internal recti muscles, and now presents well-marked ataxy of all four extremities, with slight muscular weakness, with very slight disturbances of sensibility, with swaying of the body on closing the eyes, and the failure of reflex irritability of the tendons—therefore presenting an almost complete picture of the symptoms of sclerosis of the posterior columns.

Among chronic diseases syphilis in particular has often been credited with being the cause of tabes; with what justice cannot as yet be determined. E. Schulze, in his dissertation, cites quite a series of cases in which no other cause but previously existing syphilis could be brought into etiological connection with the tabes. Judging by my own experience this connection seems doubtful, although I have often enough seen the development of other forms of myelitis in syphilitic patients. On the other hand, Fournier most recently insists on this connection as being comparatively frequent, which, if it should be confirmed, would of course be a point of the utmost importance in the therapeutics of the disease.

The connection between hemorrhoids and tabes is still more obscure and doubtful. The same is true of the causation of tabes by the *suppression of sweating of the feet*, as has been so often claimed.

E. Schulze also, in some instances, assigns a causative agency to *traumatic injuries:* fracture of the thigh, a fall upon the belly, the shock of a gunshot wound, concussion of the spinal cord, etc. This probably requires further confirmation.

Finally, various authors adduce *excessive tobacco-smoking* among the causes of tabes. We are unable to support this in itself not improbable assertion from our own experience, but neither can we deny it.

Over against all these statements, however, it must also be emphatically declared that, in no inconsiderable number of cases of tabes, the most careful investigation is able to show *no cause whatever* for the disease.

If the very existence of a causal connection between the conditions above mentioned and tabes may often be called into question, it must be admitted that we know still less with regard to the method in which this causal connection proves effective in the individual case; in what way sexual excesses or exposure, taking cold, or mental impressions, induce the disturbances of nutrition in the spinal cord lying at the foundation of the group of symptoms which constitute tabes. Of course the most varied opinions have been advanced on this subject, all of which, however, have been confined more or less to the domain of hypothesis. This much seems certain, that all cases do not have the same pathogenesis, but that those alterations which we designate as sclerosis or gray degeneration may finally be developed from various starting-points within the spinal cord. In view, however, of the great difficulties which lie in the way of obtaining anatomical material from the earliest stages of tabes, these points cannot, at the present time, be positively decided.

The views with regard to the origin of tabes which seem to us most plausible are those advanced by Remak, Sen., reproduced by Cyon, and still further carried out by Waldmann. We lay these before the reader, without, however, vouching for their correctness. According to Remak, the disease may arise in two ways. In the one, the central nervous apparatuses are primarily affected and disturbed in their nutrition by over-irritation and overexertion; primary atrophy and degeneration of the same take place, as a result of which, however, inflammation and hyperæmia may also afterwards be developed. Under this head might be ranged those cases where tabes arises from hardships and overexertion, from sexual excesses, from mental disturbances, and the like.

The other way is by the primary development of an inflammatory process in the interstitial tissue of the posterior columns, which only secondarily induces atrophy and degeneration of the nervous elements. To this category belong those cases of tabes which originate in taking cold, in traumatic injuries, the suppression of perspiration in the feet, acute and chronic diseases, and especially syphilis.

In the second group of cases, the interstitial changes lying at their foundation may either begin originally and principally in the tissue of the posterior columns, thus constituting a primary, chronic interstitial myelitis, or they may arise by extension from a chronic spinal meningitis. The inflammation of the pia mater is, in these cases, the primary step, the sclerosis of the posterior columns only secondary, extending thither from the meninges. This order of things has been especially noticed by Waldmann.

It is evident that before these views can be considered as scientifically established, pathological anatomy must demonstrate, first, that there is such a thing as a primary, non-inflammatory, degenerative atrophy of the posterior columns; second, that in certain cases of tabes this alone exists at the beginning; third, that in other cases of tabes the interstitial changes are the primary disturbance; and fourth, that the meningitic changes occasionally found in the autopsies of those who have had tabes, preceded the other changes in point of time. We shall presently see, in looking at the pathological anatomy of the subject, how far these postulates are or are not sustained.

However plausible these pathogenetic views may therefore be, they must nevertheless, for the present, be regarded as merely hypothetical.

Pathological Anatomy.

In cases taken from the later stages, *macroscopic inspection*, as a rule, enables us to recognize an *atrophy and wasting of the spinal cord*, especially in its lower divisions, and on careful observation this is also found to be especially the case in the region of the *posterior columns*, which appear somewhat *sunken* and *narrowed*.

Even on looking at it through the pia mater, or, if this is thickened and cloudy, after the removal of the same, we may recognize a *gray* or *grayish-yellow discoloration* along the posterior median fissure, at both sides of the same, extending almost throughout the entire length of the spinal cord.

As a rule, the cord shows a distinct increase in its consistency; sometimes, however, this appears quite normal, very rarely being diminished.

Generally, also, the *posterior roots* are discolored, gray, translucent, thin, atrophied, in great contrast to the normal white anterior roots; this discoloration and atrophy are marked with striking distinctness in the cauda equina in particular.

The *pia mater* usually appears cloudy and thickened, connected by numerous adhesions to the dura mater, which is but rarely thickened and roughened, the pia showing abundant bony plates and sometimes stronger pigmentation. This change is almost always confined to the posterior division of the spinal cord, and corresponds in some degree to the area of extension of the gray discoloration; more rarely the pia mater appears altered over a larger area. The *spinal fluid* always appears increased —sometimes to a large degree. The anterior and lateral divisions of the spinal cord, like the anterior roots, usually appear, on external inspection, to be quite intact.

More accurate information can only be obtained by the study of numerous *transverse sections* of the cord.

In the most well-pronounced cases, the various transverse sections show the posterior columns entirely, or throughout their greater extent, as gray, translucent, of firmer consistency, or more gelatinous, and of a grayish-yellow color. They are often wasted, sunken, evidently shrunken, so that the two posterior columns appear more approximated to each other, and show greater precipitousness in the direction of their course. This is often very marked, particularly in the dorsal portion. (See Fig. 8.)

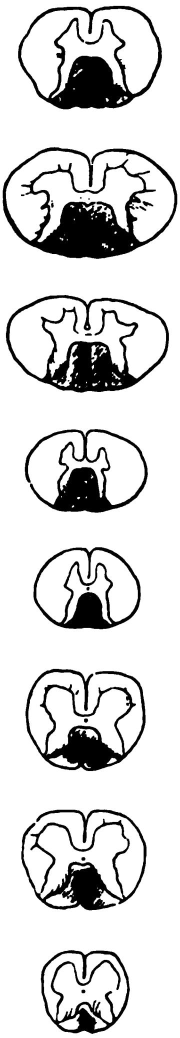

FIG. 8. Sclerosis of the posterior columns. Half diagrammatic. The most marked development is shown in the upper lumbar and the thoracic portion. In the cervical portion there is a less uniform involvement of the posterior columns.

Generally the degeneration is not uniformly distributed over the entire transverse section of the posterior columns [Hinterstränge], the middle portions and those peripheral portions lying next to the pia mater being ordinarily most intensely affected, and often still separated from the posterior commissure and the posterior horns of gray matter [Hintersäule] by a narrow zone of white substance. As a rule, nevertheless, the external portions of the posterior columns ["die Keilstränge," the "Funiculi cuneati" of Burdach. — TRANS.] are the parts, in particular, which are likewise affected, thus affording a characteristic contrast to pure, secondary degeneration which is confined to Goll's columns ["die zarten Stränge," "Funiculi graciles" of Burdach.—TRANS.].

It is also rare for the posterior columns to be the subjects of gray degeneration throughout their entire length. In the lowest lumbar division, we often see but a slight gray discoloration in the external half of the columns; this increases in width as we ascend, until finally, in the upper half of the lumbar enlargement, the entire transverse section of the posterior columns appears discolored; this then generally continues upwards throughout the entire dorsal portion, again to diminish in the cervical portion, and finally to be limited to the columns of Goll. In the majority of cases, therefore, the rule holds good that the intensity and extent of the process is greatest in the upper lumbar and the dorsal portions, diminishing both upwards and downwards from these points.

At the same time the changes may also extend upward into the medulla oblongata, into a portion of the restiform bodies, but then they cease there without any definite boundaries.

Not rarely, however, even the unaided eye can recognize a slight grayish discoloration of the neighboring posterior and external divisions of the *lateral columns*, which may extend forward as far as the anterior columns, in a gradually narrowing seam, admitting of no sharp line of demarcation between itself and the normal tissue.

In like manner the *posterior horns of gray matter* are, in many cases, also not free; they appear of a darker gray, shrivelled, distorted; the pillars of Clarke have repeatedly been found altered.

This is the picture of the fully developed, typical cases, such as most frequently occur.

At the *first beginning* of the disease, however, if anything at all is to be found, it is only a pair of narrow, scarcely recognizable gray strips at each side of the median line, in those portions of the posterior columns designated by French authors as the "rubans externes" (Charcot, Pierret). But in some cases, also, macroscopic inspection shows nothing abnormal and the microscopic alone reveals the beginning sclerosis.

In the *oldest* and *most severe* cases, on the contrary, the spinal cord, throughout long portions of the same, appears dwindled, atrophied, and hardened in its entire thickness. On making a transverse section it is found, almost throughout its entire extent, transformed into a gray, translucent mass, in which the usual picture of a transverse section is no longer distinctly to be recognized.

But complete information with regard to the kind and the extent of pathological change present can only be attained by the *microscopic examination* of hardened preparations, treated with various coloring matters.

Histologically, we may, in the *earlier stages* of the disease, at first recognize a greater or less degree of thickening of the interstitial tissue, a greater wealth of nuclei in the same, often enlarged and very much developed Deiters' cells; in the nerve-fibres there is generally not much to be seen that is abnormal, aside from simple emaciation, dwindling, and final disappearance of the same; a slight thickening of the nerve-fibres is rare; there is usually no breaking down of the medullary sheaths, nor fatty degeneration of the same, nor swelling of the axis-cylinders; it is merely a question of simple atrophy and disappearance of the fibres. Generally, especially in the more recent cases, numerous granule cells are also to be found, and these are sometimes particularly abundant in certain definite zones of the diseased tissue and scanty in others. The vessels are commonly thickened, their walls rich in nuclei. Corpora amylacea are scattered throughout the tissue in greater or less number.

In the *later stages* the principal mass of the structure is composed of a firm, fibrillar, often wavy connective tissue, which contains numerous nuclei, and is usually disseminated with innumerable corpora amylacea. Most of the nerve-fibres have entirely disappeared, ordinarily not even a trace of the axis-cylinders is to be seen; but even in the oldest cases one will still find quite a large number of single, isolated, but well-preserved nerve-fibres scattered through the increased, firm connective tissue. The vessels are sclerotic, the number of granule-cells is small, but generally there is an increased number of admirably developed, large spider cells.

The *posterior roots*, so far as they run their course within the cord in the posterior columns, endeavoring to reach the gray posterior horns, usually take part in the degenerative process; their fibres are broken down, atrophied, have grown more sparse and thinner, and are separated from one another by broader bands of connective tissue.

The microscope also generally gives the best information with regard to the more accurate localization of the process, with regard to the degree of its *extension through a transverse section of the cord.*

In the posterior columns, as a rule, the entire transverse section is more or less attacked, although to a very varying degree of intensity in different cases. Strictly speaking, however, this only holds good with regard to the upper lumbar and thoracic regions. In the lower lumbar region the middle and innermost divisions are often tolerably free; in the cervical region, on the contrary, the external divisions are generally free while the columns of Goll are principally or exclusively attacked. These then generally behave exactly as in simple secondary degeneration. The bundles of roots lying within the posterior columns are always affected likewise, and even within the posterior horns of gray matter they show all the evidences of degenerative atrophy.

It is, of course, a matter of importance to find specimens showing what part of the posterior columns is involved in the lightest cases and is the earliest attacked. The opportunity for this is, naturally enough, but rarely presented. In such cases Pierret found the external ribbons of the posterior columns (bandelettes externes) exclusively or pre-eminently attacked, the columns of Goll, on the contrary, being free (see Fig. 9, *a*, *a*).

In contrast and complement thereto, Pierret, in one case, found the columns of Goll exclusively involved in the sclerosis. The symptoms observed during life were quite different from those belonging to tabes.

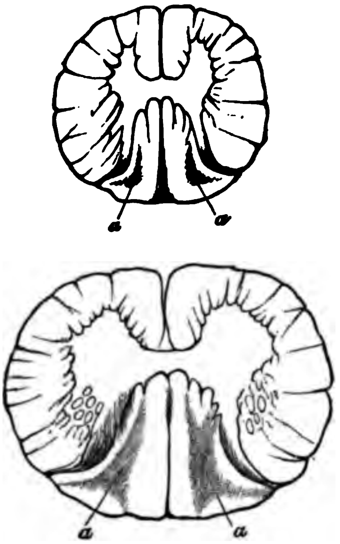

FIG. 9. Sclerosis of the posterior columns, confined to the external ribbons of the same, representing the first stage of sclerosis of the posterior columns in tabes. (*a*, *a*.) After Pierret, Arch. de Physiol. IV. Pl. 8.

The microscope furthermore reveals a regular participation in the disease on the part of the *gray posterior horns*, manifested by disappearance of nerve-fibres, thickening of the connective tissue, but very slight changes in the ganglion-cells, which at most appear somewhat more pigmented. Clarke's pillars also appear to be regularly involved, even though their ganglion-cells frequently remain tolerably intact. This involvement of the posterior horns is, according to Lockhart Clarke, so constant that he raises the question whether, after all, the posterior gray horns are not *the first* to be diseased, or, at all events, are not very early affected in all cases. This is a point worthy of all consideration.

In like manner there is also, almost always, especially in the later stages, a well-defined extension of the disease to the *lateral columns*, which is very variable and sometimes quite considerable in extent. A sclerotic stripe, varying in width, and diminishing in width anteriorly, extends forward from the gray posterior horns along the periphery of the lateral columns, sometimes even extending far into the anterior columns.

Sclerosis of the posterior columns is therefore, in the majority of cases (perhaps in all ?), *not* the only condition found in tabes, but the process extends beyond the posterior columns to the lateral columns and the posterior horns, often involving them to a very considerable extent. Such a case is given, for example, in the accompanying Figure 10.

Sometimes, also, degeneration, sclerosis, and atrophy of the ganglion-cells is met with in the *anterior gray horns*, although this is a rare form of change. According to Pierret this alteration stands in connection with sclerosis of the posterior inner root-bundles, and extends from these along the bundles of fibres that radiate into the anterior gray horns.

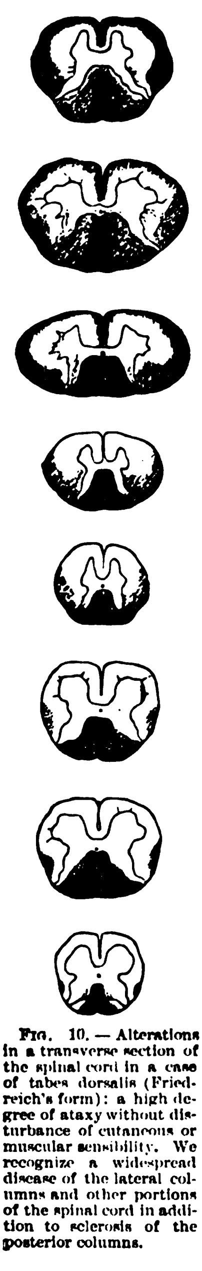

FIG. 10. — Alterations in a transverse section of the spinal cord in a case of tabes dorsalis (Friedreich's form): a high degree of ataxy without disturbance of cutaneous or muscular sensibility. We recognize a widespread disease of the lateral columns and other portions of the spinal cord in addition to sclerosis of the posterior columns.

After this brief exposition of the principal and certainly the most essential anatomical changes in tabes, various questions at once arise which are of the highest importance to the pathology of the disease, whose solution falls chiefly within the domain of pathological anatomy, but has, as yet, hardly been undertaken. What is the true essence of the diseased process ? Is it inflammation ? Is it simple degenerative atrophy ? Is it secondary degeneration ? Is the sclerosis of the posterior columns the only essential part of the process ? Where does it begin, in what portion ? How does it advance ? What is primary and what secondary about the changes ? Are not certain portions of the gray substance and of the lateral columns affected with equal regularity, and is not sclerosis of these parts of equal importance with that of the posterior columns ?

All these questions still await their solution. Many attempts have been made to answer them, and with very varying results. Conclusive proofs could scarcely be brought forward for any of the opinions advanced, and but too often the preference of a writer for a certain theory, or some preconceived opinion of his, has been sufficient ground for causing him to group or to explain the facts in a certain way.

The most recent views differ widely even with regard to the general pathological significance of the process. While Leyden treats of tabes under the head of chronic myelitis he nevertheless denies the inflammatory character of the process under consideration, and accounts for it rather as a peculiar chronic degeneration of the nerve-tracts, without any special participation on the part of the connective tissue and without inflammatory manifestations. He considers the main thing to be the atrophy of the nerve-fibres; that this constitutes the starting-point of the process, which is probably first localized in the external portions of the posterior columns.

Charcot, indeed, coincides with Leyden in so far as to locate the starting-point of the process, in all probability, in the nerve-elements (parenchymatous sclerosis); but he regards the process as a chronic inflammation, and gives prominence to the hyperplasia and fibrillar transformation of the connective tissue at the expense of the nerve-elements.

Friedreich, too, has long since declared himself of the opinion that sclerosis of the posterior columns represents a chronic inflammatory process leading to secondary atrophy of the nerve-elements.

C. Lange, on the contrary, believes that so-called secondary degeneration plays a prominent rôle in sclerosis of the posterior columns, and is inclined to attribute it to a primary affection of the roots caused by meningitis.

It certainly cannot be denied, on making an unprejudiced examination of the anatomical conditions present, that secondary degeneration does indeed play an important rôle in sclerosis of the posterior columns. The gray degeneration so commonly present in the cervical portion, confined to the columns of Goll, is very certainly only of a secondary nature, and is always found present when the greater part of a transverse section of the cord in the lumbar region is degenerated. But how and where we are to establish the boundary between secondary and primary degeneration cannot as yet be determined. Evidently, also, secondary degeneration —such, for instance, as occurs in chronic transverse myelitis—has in many cases been confounded with tabes; thus, for instance, in the two cases of Robitzsch.

At all events, most of the more recent observers are entirely agreed in the opinion that the affection does *not* begin in the posterior roots, but within the spinal cord itself. Vulpian speaks with all positiveness against the possibility of the former; Charcot and Pierret locate the beginning of the process very positively in the spinal cord itself, and Leyden is of the same opinion.

The French school has recently made the attempt, based on some fortunate post-mortem discoveries, to locate more definitely the point of the first beginning of sclerosis in the posterior columns, and to investigate the way and method of its further spread from this point of origin. The views of Charcot and Pierret, to whom we are indebted for the investigation of this difficult and important question, are essentially as follows: The sclerosis begins in the external bands of the posterior columns, at the point where the inner root-bundles mix with the vertically ascending (probably commissural) fibres of the posterior columns; this sclerosis of the lateral bands or ribbons (Keilstränge) is the only *essential* anatomical change in ataxy. The sclerosis of the columns of Goll (zarte Stränge) is to be regarded principally as a secondary ascending degeneration. At the beginning of the disease the posterior roots do not show the slightest change. In the lateral bands of the posterior columns, the sclerosis now gradually spreads upwards from root to root; from here it spreads to the columns of Goll, in part directly, and in part (and principally) as secondary degeneration; it furthermore spreads from here to the posterior gray horns, to the posterior roots, and to the external portions of the lateral columns, and finally, in rare cases, to the anterior gray horns. (The only observation which stands opposed to these statements is one published by Charcot himself, in connection with Bouchard,[1] of a case which was regarded as the beginning of tabes on account of the lancinating pains that were present, and in which the beginning sclerosis was found in the "inner and posterior" portions of the posterior columns, therefore, after all, in the columns of Goll.)

However plausible all these views may be, they can by no means as yet be looked upon as firmly established. Much still remains to be confirmed by further observation. After maturely weighing all the facts and opinions, we can probably only say this much, viz.:

It is in the highest degree probable that in tabes we have to deal with a chronic inflammatory process; that it therefore merely represents one form of chronic myelitis.

It is possible, and perhaps probable, that this chronic myelitis may take its origin in two different ways: at one time from a primary irritation and degeneration of the nerve-elements themselves (parenchymatous sclerosis), at another from a primary irritation and proliferation of the interstitial tissue (interstitial sclerosis) — thus giving a double method of origination of tabes, as was believed by Remak, Senior.

It is possible, and perhaps probable, that the sclerosis begins in the external bands of the posterior columns, and spreads from here further, and that the sclerosis of the fasciculi graciles, or Goll's columns, must, to a great degree, be regarded as a secondary degeneration.

It is certain that the disease does not begin in the posterior roots.

It is, finally, probable that sclerosis of the posterior columns is *not* the exclusive and essential change in tabes, but that a simultaneous involvement of the posterior gray horns and of certain portions of the lateral columns is constant, and, perhaps, equally essential—a statement which, it is true, finds its support more in clinical observation than in the anatomical facts now before us.

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[1] Gaz. méd. de Paris. 1866. No. 7.

After this digression, it remains for us to touch briefly on the anatomical changes to be found outside of the spinal cord in tabes.

The *posterior nerve-roots* are generally found diseased in the later stages; they are highly atrophied, flat, gray, translucent, showing degenerative atrophy of the nerve-fibres, proliferation of connective tissue, and abundant corpora amylacea. In the earlier stages, however, they are also often found quite normal; the posterior roots have never been found diseased *alone* (Vulpian).

In striking contrast thereto, the peripheral sensitive nerves are always found entirely intact, even if the posterior roots belonging to them (even to the spinal ganglia) are entirely the subjects of gray degeneration.

The *anterior nerve-roots* are intact. The *spinal ganglia* are likewise generally found entirely intact.

Some have attached great value to the examination of *the sympathetic and its ganglia*, because they sought in this the seat of the disease. This has, however, always been found intact (Carré), and where gray degeneration of the main trunk of the sympathetic and the like has been demonstrated, as in the case of Chvostek, it has certainly always been on account of a rare and accidental complication.

The *peripheral spinal nerves* have likewise generally been found intact; Friedreich alone, in one case of his, found proliferation of connective tissue, and emaciation of the nerve-fibres in the sciatic nerve.

The *muscles* are generally quite normal; they are at most simply emaciated in the later stages; degenerative atrophy of the same has been found, as a very rare complication, in individual instances.

Certain of the *cranial nerves*, on the contrary, are more frequently the seat of changes. This occurs oftenest in the *optic* nerves, which show gray degeneration—essentially the same change that takes place in the posterior columns—proliferation of connective tissue, rapid and complete disappearance of nerve-fibres, and numerous corpora amylacea. The trunks of the optic nerves are generally most intensely attacked; still, the affection may also extend throughout the entire length of the optic tract as far as to the corpora geniculata. Thus far it has proved impossible to find any anatomically demonstrable continuity of the gray degeneration between the optic nerves and the posterior columns.

In quite rare cases, alterations belonging to the class of atrophy and gray degeneration have also been found in some other cranial nerves (oculomotorius, abducens, hypoglossus).

The nuclei of these nerves, on the floor of the fourth ventricle, also appear sometimes to be affected.

Among the pathologico-anatomical changes found in other parts of the body those of the *joints* claim special interest. In those cases in which disease of the joints belongs to the symptoms of tabes dorsalis we find a disappearance of the articular cartilages, a grinding off of the articular head and of the cavity for its reception, the surfaces of which are hollowed out and rough, or ground off and flattened; a peculiar characteristic of this condition is that with the rapid disappearance of bone there is so insignificant a tendency to exostosis. In more recent cases the amount of articular fluid is materially increased, points of thickening and fungosities are found on the synovial membrane, the surrounding soft parts are more or less strongly and extensively swelled and suffused with fluid.

The changes in the *skin* (bed-sores, etc.), in the *bladder* (cystitis, ulceration, diphtheria, etc.), in the *kidneys* (pyelonephritis, interstitial abscesses, etc.), in the respiratory apparatus, the digestive apparatus, etc., are the same as in chronic myelitis. They belong to the very latest stages of tabes or are more accidental complications.

Pathology of Tabes Dorsalis.

Symptoms.

I give first the *general picture of the disease* in the ordinary typical form of tabes, which includes by far the greater number of cases, and which, in my opinion, must constitute the starting-point for all further investigations and observations. The remaining rarer forms I shall endeavor to characterize briefly farther on. In view of the importance of the malady, and the wealth and manifold character of its symptoms, this delineation must be allowed a somewhat wide sweep.

The disease almost always begins with an *introductory stage* (also designated as a precursory, prodromal or neuralgic stage), which may last for a very variable period of time, generally extending over many months or a few years—sometimes, however, over a longer period of years.

The most characteristic and almost constant signs of this stage are the *lancinating neuralgiform pains;* lively, stabbing, boring pains, shooting through a part like lightning, lasting for single days or nights, or often merely for hours; pains which may be of furious severity, generally occurring in paroxysms, and consisting of an aggregation of numerous single, often merely momentary impressions of pain; pains which often remain confined for a long time to one or both lower extremities, often changing their position within these, but which also extend farther to the trunk (very generally), and to the upper extremities (more rarely).

These pains, occurring in paroxysms, are generally regarded as rheumatic in character and treated accordingly, and are often but little regarded for years. At first they recur but rarely, at intervals of months—for instance, in the spring and fall; gradually they become more frequent and more tormenting, occur with every change of weather, especially with every considerable variation of the barometer, with rain, wind, snow, and the like, after bodily overexertion, and after mental disturbances.

They may last for years without any further symptoms being developed.

Sooner or later, however, in many cases—by no means in all—symptoms appear on the part of certain cranial nerves; above all things, *diplopia*, caused by paresis or paralysis of various muscles of the eye, especially those supplied by the oculomotorius. This diplopia may be transitory and may change its place, or it may persist for a longer time, for some months; it is but rarely that such paralyses are permanent.

Quite the contrary is true with regard to the *amblyopia*, which is of almost equal frequency, not rarely arising in the introductory stage of tabes, and which more or less rapidly develops to complete *amaurosis*, being dependent on a degenerative atrophy of the optic nerve, which is hardly ever capable of recovery.

More frequent, however, than these disturbances of the highest sense, during the introductory stage, are *disturbances of the sensibility and mobility of the lower extremities*, which, after a longer or shorter time, become constantly associated with the lancinating pains, sometimes even preceding these, or at least arising simultaneously with them.

The first disturbances are usually those of a sensory character, appearing in the form of various kinds of *paræsthesia;* such as a feeling of numbness, the sense of having wool or felt on the soles of one's feet, formication in the feet, the legs, and thighs, also on the trunk, and—what is tolerably frequent and characteristic—in the ulnar domain of one or the other hand; in other cases *the sense of a tight girdle* at various heights on the trunk, or also on the extremities, about the knee- or ankle-joint. *Anæsthesia* which can be objectively demonstrated is very rare at this time.

On the other hand, the sensations of *motor weakness and insecurity*, of growing more easily fatigued on standing or walking for any length of time, belong to the most constant symptoms. These may also, in part, be regarded as belonging to the class of sensory disturbances. That striking sense of fatigue, in particular, of which the patient is often aware even before he has left his bed in the morning, or which becomes quite oppressive on the slightest exertion, especially on standing, and which is followed by an uncomfortable disquiet in the limbs, might be regarded, in part, as an irritation or paræsthesia of the sensitive nerves of the muscles.

Undoubtedly, however, true *motor disturbances* are also associated with this condition; patients decidedly lose the strength of their limbs: exertions which were formerly borne with ease become a severe effort; their powers of endurance, in walking and standing, diminish materially. Patients find the performance of these acts increasingly difficult and disagreeable; they feel themselves unsteady on their legs, believe that they sway to and fro, or actually do so to a slight degree, and yet sufficiently so to be capable of objective demonstration. In many patients all these manifestations increase materially when it grows dark, and in such persons we can also always recognize a higher degree of disturbance when they close their eyes. All these manifestations may require a variable length of time for their development.

In many instances *disturbances of the bladder* appear sooner or later (difficulty of micturition, dribbling of urine thereafter, etc.), and almost more frequently there is *weakness and irritability of the sexual functions* (various grades of impotence, insufficient erections, premature ejaculations, nocturnal or diurnal pollutions, increased excitability on coming in contact with women, etc.).

Among the much rarer disturbances at this period of the disease are severe *attacks of gastralgia* (crises gastriques), which doubtless stand in close relation to the lancinating pains; peculiar *affections of the joints* (arthropathies des ataxiques); *head symptoms* of various kinds, as a sense of confusion, headache, pain in the neck, evidences of an oversupply of blood to the head, dizziness, great psychical irritability or slight psychical aberration, etc.

There is usually, at this period, but a very slight sense of illness; nutrition is good; there is never, or but very rarely, any fever. Finkelnburg and M. Rosenthal claim to have seen slight febrile disturbances in the initial stage.

While all the manifestations hitherto described increase, *the stage of the fully developed disease* then approaches, generally very slowly and gradually. This may, with some propriety, be called the ataxic stage, on account of the symptom which most forcibly strikes the eye. Of course it is impossible to establish a sharp boundary between the stages.

The unsteadiness of the gait increases more and more, until distinct *disturbance of co-ordination* (*ataxy*) is developed, and the patients clearly show the *ataxic gait;* their legs are moved unsteadily, irregularly, and with a flinging motion, the toes are pointed outwards and thrown into the air, the heels are brought to the ground with a stamp; the gait becomes insecure, swaying, staggering; the majority of such patients anxiously follow their steps with the eye, and can only walk with difficulty, or perhaps not at all if they turn away or close their eyes.

At the same time their powers of endurance diminish more and more; the promenades grow shorter and shorter; finally, every little trip throws the patient into a profuse perspiration; there is no longer any question of his standing on his feet continuously.

In remarkable contrast hereto, it appears, on examination in the lying posture, that *the gross strength of the legs is not at all, or but very little, reduced;* that single movements can be executed with almost normal strength and certainty. Still, on more careful examination, in the majority of cases, slight local pareses may be recognized. Tension of muscles is, however, entirely lacking.

Soon, however, well-developed ataxy of the legs shows itself, even in the lying posture. Somewhat more complicated movements (movement in a circle, the touching of an object held before them, etc.) are accomplished with increasing uncertainty, with many zigzag motions, etc., in spite of the anxious assistance given by the eye. This is frequently still more the case if the patients close their eyes, the movements then becoming quite uncontrolled, irregular, unmeasured.

In the further course of the disease the ataxy keeps increasing; patients are obliged to avail themselves of one, and afterwards of two canes; eventually they have to be led; then they can no longer walk under any circumstances, and finally they can no longer stand. On attempting to do so, swaying and staggering at once begin, or the patients fall to the ground outright.

Sooner or later *the ataxy also extends to the upper extremities;* this occurs with very variable rapidity in different cases, and sometimes does not occur at all, even where the disease has existed for years. The more delicate and complicated movements—writing, piano-playing, handiwork, etc.—are the first to become difficult or impossible; the attempt to take hold of things is accomplished with singular, jerking, irregular movements; the acts of dressing, eating, and the like become visibly more difficult, and the patients are thereby rendered to a great degree helpless.

At the same time the *disturbances of sensation* are liable to *increase;* the lancinating pains continue with varying intensity and frequency, the sense of a tight girdle is generally very considerable and sometimes increases to a feeling of oppression and constriction. The *diminution of sensibility* in the legs is now likely to be distinctly and objectively demonstrable, though to a very variable degree; sometimes it can hardly be demonstrated, even on the most careful examination, at other times it is of a high degree and well pronounced. *Partial paralysis of sensation* is also very common—analgesia, as well as partial paralysis of the sense of touch and the perception of temperature. *Retardation of sensitiveness to pain* is not rare, as well as the long continuance of sensations, or *hyperalgesia* with diminished sense of touch. Almost always corresponding *anomalies in muscular sensibility* can also be demonstrated, such as a diminished perception of the position and attitude of the extremities or of passive movements, greater difficulty in performing movements with closed eyes, etc.

The *reflex action of the skin* acts variously; sometimes it is increased, generally normal, often lowered or extinguished. On the other hand—and this seems to be a very noteworthy symptom — the *reflex action of the tendons* is always *completely extinguished*, and this seems to stand in no regular relation to the reflex action of the skin.

The *disturbances in function of the bladder and sexual organs* already referred to increase in intensity. The nutrition of the muscles generally remains intact for a very long time; the same is true of the nutrition of the skin; at this period bed-sores are very rare.

In some instances various rarer symptoms are added to the disturbances already named, whereby the picture of the disease may be rendered very manifold, but which must yet be looked upon as subordinate and by no means regular manifestations. Among these are disturbances of hearing, nystagmus, slight derangements of speech, troubles with the joints, muscular twitchings, muscular atrophy, attacks of suffocation, difficulty of swallowing, etc.

Aside from the previously described disturbances of motility, of sensation, of reflex action, etc., an objective examination reveals but very little. The spinal column is generally quite normal, nowhere sensitive. It is but very seldom that points which are painful on pressure can be demonstrated thereon. The urine usually remains normal for a long time. The internal organs are healthy. The general nutrition ordinarily remains good for a long time ; sleep, appetite and digestion are apt to be good, notwithstanding that patients suffer from more or less obstinate constipation. Many patients are strikingly well nourished, of blooming appearance, corpulent ; some, to be sure, present a pale, sickly, faded appearance.

The psychical functions, intelligence, memory, disposition, etc., generally remain intact for a long time—often permanently. The observation of Steinthal that patients sick with tabes rejoice in a striking degree of cheerfulness and contentment, and bear their pitiable troubles with wonderful good humor, can often be confirmed. Of course, this is by no means always true, and but too many patients experience the burden of their tortured existence with double weight.

Thus matters may remain in this stage for many years (5, 10, 15, or 20 years), slowly and gradually changing for the worse, and this does not exclude the existence of periods of standing still and fluctuations towards improvement. The helplessness of the patients constantly increases, their discomforts augment, the pains do not abate, and the whole misery of their condition is made more insupportable by the amaurosis, the incontinence of urine, the slowly developing vesical catarrh, etc.

And thus the *final stage* gradually approaches—the stage of true paralysis and of increasing cachexia (stadium paralyticum, paraplegic stage). The motor power diminishes sooner or later, faster or slower, finally reaching the point of complete paraplegia ; the legs grow stiff, contractions set in, and deformities caused thereby, the muscles become more and more emaciated, the extremities grow thin and skeleton-like.

Distinct disturbances of digestion now set in, the appetite diminishes, the bowels grow increasingly sluggish, and when a movement of the bowels is about to take place it can no longer be held back. The prolonged paralysis of the bladder leads to cystitis with its results; finally bed-sores set in, accompanied with fever, and if an earlier end is not put to the sufferings of the patient by some intercurrent disease (typhus, pneumonia, pulmonary phthisis, dysentery, or the like), death finally occurs through general exhaustion.

This is the way it goes in the worst cases. Sometimes, however, we see the disease, in its earliest stages, turn towards a cure; it is true this is seldom enough the case—still it does occur.

Or the disease may come to a stand-still for a longer time, the patients remain for decades of years in the same situation, without any material fluctuations, until finally the terminal stage brings about the end in a comparatively short space of time, or the unfortunate victim is carried off by some intercurrent disease.

The *analysis of the individual symptoms* of tabes brings us in contact with numerous interesting problems in the pathology and physiology of the spinal cord, which may be conducted toward their solution by the accurate clinical analysis of this disease and through careful histological examinations.

Disturbances of Sensibility.

Among these, *pain* stands largely in the foreground, partly on account of its early occurrence as regards time, partly on account of its great severity and frequent recurrence.

This is very particularly true of the so-called *lancinating pains* (boring, darting like lightning) which belong to the most regular manifestations of the initial stage, but also not rarely accompany the disease throughout its entire course.

These are the excessively severe boring or piercing pains—darting through like lightning, or sometimes raging in one spot for some seconds, and occasionally conveying the sense of a tight cord—which have already been graphically described by so many authors, and which we have also already previously attempted to characterize (p. 75).

They usually occur in paroxysms, which are repeated with varying frequency, and which themselves, again, are composed of a larger number of single painful sensations. They are at times confined to a small, circumscribed region of the skin, and are then not rarely accompanied with a high degree of circumscribed hyperæsthesia at the same spot, causing a light touch to be uncommonly painful, while, on the contrary, stronger pressure appears to be grateful. Or these pains may be situated deep within the soft parts or in the bones, and one sees the patient every moment or two twitching in body and his face distorted with pain, sometimes even moaning and whimpering. Or the pains may follow certain definite nerve-tracts, in the form of neuralgia, as darting pains through the limbs, generally designated as "rheumatic." Then during the paroxysms the nerve-trunks are likely to be sensitive to pressure.

The intensity of the pain is very variable in different cases. Some patients are so little troubled with it that it requires careful questioning to determine the existence of the pain. Others are obliged to endure the most fearful tortures during the attacks, which equal in intensity the worst neuralgic paroxysms. Almost every patient gives a somewhat different description of the pain; but its momentary, boring, paroxysmal character, like a streak of lightning, almost always stands forth so prominently that it can readily be recognized.

The duration of the individual attacks is very variable: often but single darts of pain arise; at other times an attack of half an hour is all that is experienced for one or several days; but frequently the attacks last for hours and days, and generally attain their greatest intensity during the night. The intermissions may be quite free from pain, or may present only moderately painful sensations.

The frequency of the attacks is extremely variable: sometimes they are repeated in the course of days or weeks; at others the individual attacks are separated by intervals of months, and even of years.

It is not to be denied that external impressions are of the greatest influence on the occurrence of the attacks. This is undoubtedly true with regard to the influence of the weather. Although this influence may not be capable of being demonstrated with certainty in all patients, yet it certainly exists in many, and I have often been surprised at the unanimity with which my tabes patients complained of attacks of pain on certain days. In our climate, patients have the most reason to fear wind, a low barometer, rain and snow, also fog; hence, doubtless, the often observed greater frequency of the attacks in the spring and autumn. In the height of summer those days are especially to be feared on which there are thunder-storms.

Aside from the influence of weather, the attacks are also sometimes brought about by bodily exertion, emotional disturbances, the act of coition, and the like.

The lancinating pains undoubtedly belong to the most constant symptoms of tabes; Topinard states that they were wanting 22 times in 104 cases; in 60 of my own observations, in which a statement is made with regard to them, they were absent only 8 times; on paying more particular attention thereto, they may perhaps be found still more frequently. Quite lately it happened that one of my patients, on being repeatedly questioned, stated that he had never had any pain; and then, when he happened to have a characteristic attack of pain, declared that that was his old "rheumatism," from which he had suffered for many years. Generally the pains precede all other symptoms for a longer or shorter time; sometimes, however, they occur at the same time with other symptoms, or follow them. Whenever the characteristic attacks of pain are met with, we may seriously suspect the presence of tabes; at the same time, it would be a matter of interest to determine more positively whether similar pains may not also occur here and there without any reference to sclerosis of the posterior columns, or whether they are, as it almost appears, well-nigh pathognomonic of tabes.

With regard to the actual pathogenesis of these lancinating pains, we have as yet only conjectures, the most probable of which is that they are caused by irritation of the posterior root-fibres within the posterior columns. Charcot, with all positiveness, places the anatomical location of the same in the external bands of the posterior columns which contain the internal root-fibres, claiming that the lancinating pains are entirely characteristic of sclerosis of these bands.

According to this, they might probably also occur in other diseases of the spinal cord as soon as these involved the above-named bands—for instance, in multiple sclerosis.

Analogous perhaps to the lancinating pains are the visceral neuralgias described of late by a number of writers: the urethral and rectal colics, the gastralgic attacks (Delamarre, Charcot), the attacks of renal colic (Raynaud), etc., to which we shall refer again farther on.

Pain in the back belongs decidedly to the rare symptoms of tabes. In many cases patients do not complain of the least abnormal sensation along the vertebral column; this is nowhere sensitive to pressure or on motion. Sometimes complaint is made of circumscribed or more diffuse pain in the vertebræ, between the shoulder-blades or in the small of the back. But these are manifestations of only slight severity. In some cases, points which are painful on pressure may be found on the spinous or transverse processes of single vertebræ—a fact to which Mor. Meyer has recently again called attention[1]—which furnish an important basis for treatment, but which are, unfortunately, only too rare. Whenever there is prolonged and severe pain in the back, when this pain is materially increased by movements of the body or of the extremities, by riding over a rough road, by pressure on the spinal column, we may suspect the immediate cause to lie in meningitic complications, as has also been plainly shown by the descriptions of Waldmann.

Among the varieties of *paræsthesia*, which rarely fail in the picture of tabes, the most prominent is *the sense of a tight girdle.* It is apt to be extremely annoying to the patient; sometimes it appears as a very troublesome sense of being tightly laced around the thorax or the abdomen, combined with a more or less severe sense of oppression, or it appears to the patient as if he were enclosed in a tight coat of mail. But it also not rarely occurs on the extremities, at the knee- or ankle-joint, or like a garter below the knee. The intensity of the sensation is very variable, fluctuating from one day to the next, but rarely rising to the point of severe pain. The most probable views with regard to its pathogenesis have already been given on page 75.

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[1] Berl. klin. Wochenschr. 1875. No. 51.

The statements of patients with regard to the presence of other varieties of paræsthesia, which so often occur, especially in the lower extremities, are exceedingly manifold. The most common are the feelings of formication, of numbness, and of insensibility. One patient says that he feels as if he were walking on felt soles or on wool, another as if he were walking on bladders filled with water, etc. A sensation of burning in the skin, or of cold, is also not rare. The feeling of formication is not rarely confined to circumscribed spots in the skin or to the domain of single nerves, as to the thigh, the perineum, or anus, etc. This or a similar abnormal sensation is also especially frequent in the ulnar region of the upper extremities, and I must, according to my experience, agree with Erdmann when he ascribes a certain significance to this symptom, which so often occurs in the initial stage of the disease.

These forms of paræsthesia belong to the most common manifestations of tabes, and we shall rarely find a case in which the one or the other cannot be demonstrated. They usually appear in the earliest stages, and may then change their seat and the area of their extension with the progress of the disease. At the same time there is not much that is characteristic about them, as they occur in so many diseases of the spinal cord. We have already expressed ourselves with regard to their pathogenesis on page 74.

The appearance of *hyperæsthesia of the skin* is much more rare in tabes. Still, it occasionally takes place to a high degree, entirely irrespective of the quite circumscribed hyperæsthesias which sometimes accompany the lancinating pains. Then large regions of skin may possess a special sensitiveness over against certain kinds or all kinds of irritation. I have seen a man who showed a high degree of hyperæsthesia towards impressions of temperature, over the entire back, while at the same time there was anæsthesia towards impressions of touch. It is also not rare to see a moderate paralysis of the sense of touch accompanied by a high degree of hyperæsthesia towards impressions of pain.

The statements of physiologists with regard to the occurrence of hyperæsthesia in lesions of the posterior columns would sufficiently explain this manifestation; only one would then expect hyperæsthesia to be much more common in tabes. (Compare page 73.)

Anæsthesia of the skin certainly belongs to the most regular manifestations, and to those most carefully studied with reference to the theory of the ataxy. The possibilities with regard to the occurrence, the intensity and the extent of this disturbance, are positively innumerable. Almost every case behaves differently in this respect.

The patients themselves are often unconscious of the anæsthesia which objective examination demonstrates in them. Generally, however, they are informed on the subject by means of various little discoveries which they have made on themselves: they no longer feel the floor distinctly; articles which they touch seem to them to be covered with velvet; they cannot judge with certainty of the temperature of a foot-bath; they cannot hold on to small objects with their fingers if they turn their eyes away; they can no longer button their clothes, or tie a cravat, without the looking-glass; they cannot readily handle things in their pockets; and more of the same sort. If there is a higher degree of anæsthesia, the patient is in great doubt, when in the dark, as to the existence and position of his legs, and he knows that the anæsthetic parts must be taken hold of vigorously before they recognize any sensation.

A more accurate picture of the kind and the degree of anæsthesia can, however, only be obtained by an objective examination. With regard to the methods to be employed in carrying this out we have expressed ourselves in detail elsewhere,[1] and would refer the reader to what is there said. By these means we can, first, recognize the most varied grades of *dulness of sensibility*, often such insignificant and circumscribed disturbances—especially on the soles of the feet, the toes, and back of the feet—that they can only be demonstrated by the most careful examination. Generally, however, we find distinct and easily demonstrable diminution of sensation, most marked in the feet and legs, but also not rarely extending over the thighs, the trunk, and even portions of the upper extremities. The higher degrees of cutaneous anæsthesia—such as occur, for instance, so frequently in transverse myelitis, in slow compression of the cord, etc.—are but seldom observed in tabes; even in the latest stages such severe anæsthesia is an exception, and in the earlier stages, even though extending over many years, it may be stated as a rule that the cutaneous anæsthesia only reaches a moderate grade.

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[1] *Erb*, Diseases of the Peripheral Nerves. This Cyclopædia, Vol. XI. p. 212 and following.

On the other hand, especially in the later stages, more careful examination very often reveals the existence of *partial paralyses of sensation.* It would carry us too far to describe in detail all the individual possibilities of these interesting disturbances, almost every case behaving differently in this respect. Suffice it to say that all possible combinations of partial paralysis of sensation, such as we have characterized on page 199 of Vol. XI., occasionally appear—for, is not tabes the very richest field of observation for these disturbances? Perhaps *analgesia* occurs the most frequently—loss of the sensibility to pain with retained sensibility to touch; but the reverse is also not rare: retention or even increase in the sensibility to pain, with diminution of sensibility to all or to some varieties of touch; partial paralysis of the sense of touch may be combined with analgesia or hyperalgesia, or with hyperæsthesia towards impressions of temperature, and the like. According to Topinard, sensibility to temperature is, in many cases, retained an unusually long time; in short, the individual cases present an inexhaustible variety of manifestations.

At a later period it is not rare to find a distinct *retardation of the conduction of sensations*, especially of impressions of pain, as we have already more explicitly set forth at page 70. This manifestation, which has long been known, has recently been more accurately studied (E. Remak, Osthoff, Naunyn, Hertzberg); it is not rare in the typical cases, as taught in the schools, and may even be found present in the early stages of the affection. According to the most recent observations, it at first seemed as though this retardation extended *only* to the sensation of pain, a fact which would stand in admirable accord with physiological conditions discovered by Schiff; indeed, this is by far the most frequent state of things. And the statement of patients with regard to the double sensation produced, for instance, by the prick of a needle, on account of the normal rapidity with which the sense of touch is conveyed, and the retardation of the sense of pain, is often very striking, being the most so in typical cases of tabes, and being more frequently found, on careful examination, than was hitherto supposed. But this retardation is by no means always limited to the sensation of pain; in fact, Hertzberg has demonstrated that in some cases the sensations of touch and of temperature are also retarded, although to a far less degree than those of pain.

Usually, in such cases, a corresponding *retardation of reflex action* can also be demonstrated; likewise, as a rule, a strikingly *long duration of*, not rarely a *supplementary increase in*, *the sensation of pain*, so that patients declare the highest degree of pain not to be reached until several seconds after it has begun. Intimately connected with this is the inability to separate with certainty and to count several tactile impressions rapidly following one another. These manifestations, too, have previously been spoken of.

Not less frequent than the disturbances of cutaneous sensibility in tabes, are the *anomalies of muscular sensibility*, which, long since known and acknowledged, have recently been brought more into the foreground by Leyden. Here, too, the conditions may vary much.

First of all, it may be said that the extreme *feeling of fatigue*, which is so frequent and so annoying at the beginning of the disease, may perhaps be nothing else than a perverse condition of muscular sensibility, a paræsthesia of the sensitive nerves of the muscles. Spaeth has endeavored to establish this view; I must confess that it appears to me quite plausible, notwithstanding that it is thus far incapable of direct proof. In the same category with this sensation we might probably also rank the annoying feeling of disquiet in the limbs which arises on sitting or lying still for any length of time.

But a much more frequent and important manifestation is that of *diminution of muscular sensibility* which can, by means of the proper examination, be shown to exist in persons suffering from tabes.

It is here less a question concerning so-called muscular sensibility, or electro-muscular sensibility[1] (though this may likewise be diminished), or concerning the so-called sense of power [Kraftsinn], which, as Leyden has shown, does not figure so prominently here, being an essentially psychical act, than it is a question of the diminution of what is called muscular sense [Muskelsinn], which can very often be demonstrated in patients with tabes. Such patients more or less entirely lose *the sense of the position or attitude of their lower extremities;* in the dark or with closed eyes they do not know where their legs are, how they lie, whether they are crossed, or how far apart from one another they are. If one leg is passively placed in a certain position and the patient is asked to put the other in the same position, he cannot succeed in doing this at all, or but very imperfectly, while a well person can do it with the greatest precision. These patients have likewise *lost control over the extent and the direction of the movements required of them*, if they are to execute these in the dark or with closed eyes. Their movements are thus rendered uncontrolled, excessive, shooting far over the mark or wide of it. This is generally mistaken for an increase in the ataxy which is commonly present. On careful observation, however, it is easy to recognize that the true ataxic disturbances of motion are not essentially changed, that it is not the uncertainty of movement that is increased, not the *zigzag* character of the gait when the eyes are closed, but merely that the patient is uncertain with regard to the *direction* of the motion to be undertaken, and does not know when he has reached the *limit* of the required movement. These two things are to be sharply distinguished from one another.

Finally, such patients have more or less completely lost the *sensation which judges of passive movements of the limbs.* If the appropriate tests are made, according to the method given on page 67, it will be found that much more extensive movements are required than in a well person in order to produce any sensation thereof, and that patients are very much mistaken with regard to the direction and the degree of the movements. In fact, it will be found, in the severer cases, that in certain joints such passive movements cannot be felt at all, and that, when their eyes are closed, patients are in absolute uncertainty as to the position in which their limbs have been placed.

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[1] Compare Vol. XI.

All these disturbances sometimes, though much more rarely, occur in the upper extremities as well.

It does not need to be specially insisted on, that it is not the sensitive nerves of the muscles alone which are to be held responsible for these disturbances, but that the sensitive nerves of the joints, the tendons, ligaments, fasciæ, and in part of the skin also, must take part, if the group of disturbances above described is to be brought about.

So far as concerns the occurrence of anæsthesia in tabes, the statements of different authors widely disagree. While Leyden regards anæsthesia (cutaneous and muscular) as a very constant symptom, and is even inclined to believe in its existence where good and reliable observers have been unable to demonstrate it, a large proportion of other authors are of the opinion that while cutaneous and muscular anæsthesia belong to the very frequent, they are by no means among the constant symptoms of tabes. Topinard, for instance, in 109 cases, found cutaneous anæsthesia 76 times distinctly, 15 times very lightly, and 18 times not at all; and among 50 cases that contained statements on the subject, muscular anæsthesia 20 times to a high degree, 8 times slightly, and 22 times not at all. Friedreich has repeatedly and quite recently described cases of intense sclerosis of the posterior columns in which the most accurate and exhaustive tests of sensibility, in all directions, have failed to show the slightest trace of cutaneous or muscular anæsthesia, at least during very long periods of the well-developed disease. I too must express the conviction, founded on my own experience and special investigation directed to this point, that—though they may be rare—*there are cases of well-pronounced tabes in which every sort of disturbance of cutaneous or muscular sensibility is lacking*, or, at all events, cannot be recognized on the most careful investigation, while in such cases the highest degree of ataxy may be present.

In the remaining, much more frequent cases, which certainly constitute the rule, the anæsthesia appears at very variable periods in the course of the disease. It is rare for a distinct, objectively demonstrable anæsthesia to be present in the initial stage; it generally does not appear till the beginning of the second stage. Topinard asserts outright that the cutaneous anæsthesia always appears simultaneously with or *after* the ataxy, never before it—a declaration which we cannot entirely subscribe to. During the further progress of the disease the anæsthesia may gradually increase in intensity and extent, so that, during the later periods, at least the feet and legs below the knee may become almost completely anæsthetic. Still, this is subject to the greatest variation in individual cases.

Notwithstanding the assertion of Leyden that, on careful examination, a dulness of cutaneous or muscular sensibility, or of both, can regularly be demonstrated, corresponding to the degree of ataxy present, I must, on the strength of my own observations, subscribe to the opinion of numerous other observers: that, *in patients who have tabes, there is not even an approximatively constant relation between the degree of ataxy and the degree of cutaneous or muscular anæsthesia.* In the typical and more frequent cases, it is true, both forms of disturbance are usually very well marked; but, aside from these there are numerous other cases of tabes in which there is a high degree of ataxy and only a very slight disturbance of sensibility, and still others with well-pronounced disturbances of sensibility and but very slight ataxy.

The question of the pathogenesis of all these disturbances of sensibility, as regards the anatomical basis of the same, is generally answered on the supposition that the gray degeneration of the posterior columns and posterior roots is abundantly sufficient to explain the disturbances of sensibility here found. But the proof of this is not adduced. Serious doubts on the subject are aroused by the not unfrequent cases in which the highest degree of gray degeneration of the posterior columns has been found, without any considerable disturbance of sensibility; by the experience of secondary ascending degeneration of the posterior columns; by the physiological facts, which indicate with all certainty that a lesion of the gray matter of the cord always lies at the foundation of retardation in the conduction of sensations; by the investigations of Woroschiloff, which show that the lateral columns take an essential part in the conduction of sensory impressions. This is not the proper place further to enlarge upon and to defend these doubts; but it does appear proper to state that gray degeneration of the posterior cord does not seem to us to furnish, with certainty, sufficient ground for the disturbances of sensibility in tabes. We are more disposed to conjecture that wherever there are profounder disturbances of sensibility, retardation of conduction, etc., the gray substance (posterior horns) prominently participates in the anatomical changes. This point, however, as well as the degree of possible participation of the lateral columns in the causation of sensitive disturbances, must be left to be determined by further careful investigations.

It may be permissible here to treat at once of a symptom which seems to us to stand in intimate relations to the disturbances of sensibility, and which, by the frequency of its occurrence, and through the authority of Romberg, has acquired great importance in the clinical history of tabes, viz., *the patient's swaying and falling to the ground on closing his eyes.* This belongs to the most constant and regular symptoms of tabes. Still, there are exceptions even to this; cases in which, with the highest degree of ataxy, there is no swaying on closing the eyes, or at least the swaying which does exist is not aggravated thereby; there are always cases in which there is an entire absence of any disturbances of sensibility. We have already spoken of this manifestation in detail previously (page 93), and endeavored to demonstrate its intimate connection with the disturbances of sensibility. This symptom will never be found lacking where there are distinct disturbances of sensibility in the lower extremities. To be sure it also occurs, at least in its lightest forms, in not a few cases in which, as yet, no disturbance of sensibility can be objectively demonstrated. We are therefore disposed to regard swaying on closing the eyes as the first and lightest symptom of a diminution in the so-called muscular sensibility, which is not, as yet, otherwise objectively demonstrable. Inasmuch as constant sensory impressions coming from the periphery are necessary for the maintenance of the equilibrium and the position of the body in space, it is just in this very disturbance of equilibrium (in swaying) that the least disturbance of these centripetal currents is noticed the soonest. At all events, we must adhere to the opinion that this symptom does not stand in closer relation to the symptom of ataxy.

Disturbances of Motility.

The most important among the symptoms belonging here, and the one most thoroughly discussed, is unquestionably the *ataxy*, the *disturbance of co-ordination, of tabes.*

The characteristics of this disturbance of motility are that the certainty and precision of movements, especially of all combined and complicated movements, is naturally diminished, while the strength of individual movements and the certainty of the simplest movements is not at all, or but very slightly diminished. What we have here is a disturbance in the co-ordination of movements, and not a true paralysis. Notwithstanding the frequency with which an unmistakable diminution even of gross motor power, and especially of endurance, can be demonstrated in tabes, still this paresis is by no means the typical feature in the picture of the disease. In all typical cases the true disturbance of co-ordination certainly stands very much in the foreground as compared to the paresis, and the amount of the disturbance of co-ordination is out of all proportion greater than that of any paralysis that may be present. *Ataxy, and not paralysis, is the essential and characteristic disturbance of motion in tabes.*

The method of appearance of the ataxy is, in the great majority of instances, the same. The disturbance almost always begins in the *lower extremities.* In the *lightest grades* the experienced eye notices a certain unsteadiness and irregularity in the gait; the steps grow somewhat uneven, the position of the feet somewhat irregular; more difficult movements betray a higher degree of insecurity; rapid swerving to the right or left, quick turning around, are accompanied with distinct swaying;

the patient finds greater difficulty in running and hopping, climbing stairs, stepping upon a stool, walking on a crack, walking on a waxed floor, etc.; not unfrequently they complain of an aggravation of all these troubles at evening, in the dark, or when they shut their eyes. No material disturbance is as yet noticeable in standing; only in standing with closed eyes is there a slight degree of swaying, the patient quickly grows uncomfortable, or slight twitching movements of the anterior muscles of the leg may set in. Standing on one foot grows very uncertain, and soon impossible. On lying down, however, all movements generally seem as yet altogether quiet and secure, and are commonly also performed without any material diminution in gross strength.

Soon, however, the disturbance reaches a *higher grade;* now the movements of walking grow quite unsteady, excessive, and are executed with a flinging motion; the toe of the foot is flung forward and outward with a brisk motion, the heel is brought to the ground with a stamp, the knee is held as stiff as possible, and generally the patient is seen to follow the movements of his feet sharply with his eyes, his glance directed to the ground. The gait is thereby rendered to a high degree shaky, insecure, reeling, the steps quite uneven and jerky; the line pursued in walking is irregularly zigzag.

The disturbance now also makes itself more distinctly noticeable in standing; patients sway and oscillate to and fro, especially if they are made to stand with their feet together; backward movements of the legs and trunk disturb the equilibrium; that form of ataxy has been reached which Friedreich has of late very appropriately designated as *static ataxy*, and has set up over against locomotor ataxy. The former is made manifest during the quiet fixation of the extremities or the trunk, such as is required for the quiet stretching out of an arm, the quiet holding up of a leg, or in sitting and standing; the latter occurs during the execution of movements of the trunk or extremities, and represents a lighter grade of ataxy; static ataxy appears only in the later stages and in advanced disturbances.

But now, even movements performed when lying down, and which are comparatively simple, also become distinctly ataxic, and it is precisely the study of these movements which I consider as especially calculated to insure a correct insight into the true essence of ataxic disturbances of motion. The patients are unable to touch an object held before them by means of a quiet and uniform movement of the tip of the foot, but the line of motion executed is an irregular, zigzag line; simple raising of the leg occurs in a similar line, rendered irregular by brisk side movements; the leg which has been lifted falls back upon the bed at a point more or less remote from its former position; instead of describing a circle with the tip of the foot, as the patient intends to do, he describes quite an irregular figure with a zigzag outline; it thereby becomes evident that the direction of movement in general is retained, but that a straight or uniformly curved line has become an irregular and zigzag line. Patients are unable to execute any quiet and steady movements, and even the simplest motions are executed in a jerky and abrupt manner. In many cases this becomes much more distinct if the same movements are required to be made with closed eyes; but this is only true when there is a simultaneous disturbance of sensibility, especially when there is muscular anæsthesia; to the ataxy there is then added uncertainty with regard to the direction and the degree of motion required, which thereby becomes quite excessive and uncontrolled. In those cases, however, in which sensibility is entirely intact, it is easy to demonstrate that the ataxy is either not at all influenced by closing the eyes, or but very slightly increased. And in all cases that are at all extreme it can be shown that the most careful control on the part of the eyes is *not* capable of doing away with the actual ataxy of motion.

In the *highest grades* of ataxy, finally, it becomes entirely impossible for patients to stand or walk. The legs find no support, slip out in every direction; left to themselves such persons at once fall to the ground; if they are allowed to make attempts at standing or walking, supported on either side, their legs fly around in the air, widely and confusedly, are thrown in this and that direction by all kinds of irregular, paroxysmal movements not subject to the control of the will, and are incapable of giving the least support to the body. In wonderful contrast hereto, even in these cases, is sometimes the strength with which certain movements can be executed, and the energy with which limbs, which are utterly useless for standing or walking, can offer resistance to attempts at passive motion. In spite of this, such patients are utterly helpless and are condemned to a permanent sitting or lying posture.

The ataxy is liable to appear much later in *the upper extremities* than in the lower; it seldom attains such a high grade here, and—on account of the different method of using the upper extremities—appears in a somewhat modified form. In the more frequent typical cases of tabes, ataxy of the upper extremities belongs to the more rare and certainly to the very late manifestations; just the contrary appears in those cases belonging to the group described by Friedreich; in these, ataxy of the upper extremities appears very soon after that of the lower, or almost simultaneously therewith.

It manifests itself earliest in the innumerable more delicate acts in which we use our hands: in writing, playing the piano, tying a cravat, buttoning clothing, drawing, cutting, etc. All these movements become more and more uncertain, difficult, irregular, the more so if they have to be carried out without the guidance of the eyes. At a later stage the simpler movements also become impeded, irregular, ataxic; here, too, it again appears that the disturbance consists essentially in the transformation of rectilinear directions of movement into movement in irregular, zigzag lines. If these patients are asked to touch an object held before them, with the tip of one of their fingers, they will shoot past the mark, and only reach it after repeated, irregular swaying to and fro in front of it. This is particularly marked if they are required to hold their two forefingers at a considerable distance apart and then bring them together with tolerable rapidity so that the two tips shall touch one another; here the ataxy stands forth in a striking manner. Not less characteristic is the disturbance of co-ordination in reaching for an object held before them; this is only reached by a roundabout way and with jerky interruptions; at the moment when the fingers are about to reach it they are spread out again, and the whole act of grasping is performed in an uncertain, blind sort of way; finally, in attempts to draw figures in the air with the forefinger, a circle, numbers, a cross, etc., the ataxy also stands forth very prominently—all the outlines are irregular and zigzag. This, too, becomes more so on closing the eyes, but only when disturbances of sensibility are present at the same time.

At a later period there may also be static ataxy in the upper extremities, as it has been strikingly depicted by Friedreich. Patients can no longer hold their arms still when stretched out horizontally, without the occurrence of slight backward movements of the same and of the hand; they can no longer exert uniform pressure with their hands, etc.

In the highest grades, finally, the hands become entirely useless for all the acts of daily life; patients can no longer dress themselves, can no longer carry a spoon or glass to their lips, they can hold nothing—in short, they are as good as completely deprived of the use of their upper extremities, although here too, as a rule, the muscles are still for a long time capable of a surprising development of strength.

But the ataxy does not always confine itself to the extremities. Occasionally that harmonious working together of the muscles of the trunk is also disturbed which is requisite for maintaining the balance in standing and sitting, and the body makes irregular swaying movements; ataxy of the muscles of the neck disturbs the quiet position of the head, which falls into irregular shaking and swaying. Thus in the cases described by Friedreich.

The same observer has formerly, as well as recently again, described *disturbances of speech and of the movements of the eyes* which may, without forcing things at all, be regarded as ataxic, and the existence of which has been established by Friedreich in a whole series of cases. The disturbance of speech betrays itself first as a somewhat indistinct pronunciation of words; afterwards in the form of an irregular, stuttering interruption of speech. It decidedly does not take the form of deliberate measured speech, for often whole sentences are uttered rapidly and then there is a slight stuttering, and this is repeated in quite an irregular manner, while at the same time the voluntary movements of the tongue and lips are apparently quite undisturbed. In the highest grades this disturbance may become so extreme that speech is almost incomprehensible on account of the bad articulation.

Ataxy shows itself in the movements of the eyes as a form of nystagmus, which appears on attempting to fix the eye on an object held at rest, or to follow a moving object; a nystagmus which is independent of disturbances of vision and affections of the refracting media, and which is entirely absent when the eye is at rest and no attempt is made to fix it upon an object. The greater and more numerous the movements requisite for fixing the eye on and following an object of vision, the more distinct and pronounced will be the appearance of the nystagmus, in the form of twitching movements of the eyeball, which do not follow one another with the rapidity of these movements in ordinary nystagmus, and which take place principally in the transverse direction, but occasionally also in every other direction. We cannot but concur entirely in Friedreich's designation of these movements as being ataxic; they differ from the movements of ordinary nystagmus partly by their being independent of diseases of the eye, partly by the fact of their following one another with less rapidity, and partly in their occurrence exclusively during movements of fixation.

The occurrence of these ataxic disturbances in the cranial nerves certainly constitutes one of the greatest rarities in the clinical picture of tabes; as far as our knowledge goes, thus far, it appears to be confined to the peculiar cases of sclerosis of the posterior columns described by Friedreich, and which will be given more fully farther on. In the typical forms of tabes these disturbances seem to occur rarely or never, in characteristic contrast to the conditions obtaining in multiple sclerosis, as set forth in a previous section.

But little is known with regard to ataxy in the domain of the other cranial nerves, especially the facial nerve, in tabes. In some few cases grimacing movements of the face have been observed. But at all events these are amongst the greatest rarities.

After the description given above, and if one has once carefully observed a series of tabes cases, it will not be easy to confound ataxy with any other disturbance of motion. Nevertheless in some instances difficulties may arise. Some cases of *chorea*, in particular, may easily be mistaken therefor, and yet this is because superficial observation sometimes reveals a remote similarity between the two forms of motor disturbance. For more accurate observation at once shows striking differences between them. Let any one read the admirable description of choreic movements given by von Ziemssen (this Cyclopædia, Vol. XII.), and he will not doubt for a moment that it is here by no means a question of ataxy, but true cramp. Friedreich has again recently drawn a sharp line of distinction between the two forms of motor disturbance: ataxy appears only during voluntary movements, and ceases during rest; choreic twitchings continue also during complete rest of the body, and they only incidentally disturb the otherwise quite well co-ordinated voluntary movements; choreic twitchings almost always affect also the face and the tongue, ataxic disturbance does this but seldom, and then only in a subordinate manner. We would add that in ataxy a certain definite movement (taking hold of things, walking, etc.), whenever it is repeated, is always disturbed in about the same way, while choreic twitchings have something entirely accidental and irregular about them, and form a picture of motor disturbance of a constantly and strikingly changeable type. Of course, the other accompanying manifestations would also not readily permit us to confound ataxy with chorea.

Nor will ataxy be easily mistaken for the *tremor of multiple sclerosis* to which we have already explicitly referred (page 100). In this tremor there are regular oscillations in the line of movement; in ataxy there are quite irregular zigzag movements, which by no means make the impression of a tremor. Still, it must not be forgotten that both disturbances may occur alongside of one another, as is, indeed, not rarely the case in multiple sclerosis.

Less likely yet is ataxy to be confounded with *the tremor of paralysis agitans.* The very manner of this tremor, which moves in very small oscillations, positively forbids this mistake. Added to this is the fact that the patient afflicted with paralysis agitans can, for a very long time, still execute voluntary movements with great precision and without trembling, while ataxy only arises during voluntary movements, and is absent during rest.

The appearance of ataxy almost always occurs at a period of time more or less removed from the beginning of the disease, after having been preceded by the symptoms of the initial period for months and years, sometimes many years. Indeed, we are ordinarily accustomed to reckon the beginning of the second stage from the time of the appearance of ataxy. Still, this varies in different cases; the disturbance arises with very varying rapidity, and ataxy may even figure among the earliest symptoms, or even be the first and for a long time the only symptom of the disease, as in several cases described by Friedreich. But for the ordinary typical cases the rule holds good that ataxy does not appear until after various initial symptoms have existed for months or even for years. It must still be regarded as questionable whether the cases of "acute ataxy," described by a number of observers, or those ataxic disturbances observed after acute diseases (diphtheria, typhus, variola, etc.), and which are generally developed with striking rapidity, are really to be accounted as true tabes.

Great difficulty has for a long time been experienced in arriving at a *pathogenetic explanation of the symptom of ataxy.* Innumerable attempts at an explanation have been set up, the most various hypotheses have been defended with more or less of spirit and of good fortune, and discussions with regard to the "theory of tabes" have hinged only too much on the theory of the symptom, "ataxy." It would lead us much too far to enter into a full discussion and explanation of these interesting points of dispute.

We have already at a previous place (p. 83 et seq.) entered somewhat minutely into this question, and have endeavored to make clear the method of origin of spinal ataxy. After maturely weighing the various views, we came to the conclusion, based upon the facts thus far ascertained and supported by numerous investigations of our own, *that the ataxy, in tabes, can by no means be made dependent upon the disturbance of sensibility which may happen to coexist, but that it must rather depend upon a disturbance of co-ordinatory tracts lying within the spinal cord; that it is therefore a "motor" ataxy.*

To-day we have but little to add to the explanations there given, and we have nothing to take back of the views there expressed, in spite of the explicit defence which has, in the meantime, been put forth by Leyden, whereby he endeavors to uphold his former theory. On the contrary, we are only confirmed in our opinion by renewed study of the literature of the subject and accurate observation of new cases, so that we have no doubt that the ataxy in tabes is not at all, or at least in but a very unimportant manner, influenced by the disturbance of sensibility.

In the meantime Friedreich has also opened up the question anew in a thorough and admirable work, has refuted Leyden's view, and has thoroughly fortified his own opinion, which is to the effect that the immediate cause of ataxy *does not* lie in a disturbance of sensibility, but that it must rest upon a disturbance of co-ordinatory tracts within the spinal cord.

It only remains for us here to introduce to the reader a few of the most prominent advocates of different views, so as briefly to render him familiar with the various attempts at the explanation of ataxy. In so doing, we can only permit ourselves a very brief criticism of the same.

A whole group of authors, being led thereto by the anatomical conditions found, which in the main show disease of the posterior roots and the posterior columns, and by the physiological investigations which assign to these parts only sensitive functions, or at least only the function of centripetal conduction, seek the causes of ataxy in a disturbance of the sensitive functions, or at least in a disturbance of the centripetal excitation which is conducted through the posterior roots.

A part of these authors seek the causes of ataxy, independently of the disturbances of conscious sensation, in *a disturbance of the reflex activity within the spinal cord.* Thus, according to Jaccoud, the ataxy depends in part upon a disturbance of the cutaneous and muscular sensibility, and represents an anomaly of spinal motor irradiation and of spinal reflex action.

According to Carré, ataxy is independent of conscious sensibility, but dependent upon disturbances of reflex activity, upon conscious sensibility.

Cyon regards co-ordination as taking place through a reflex process within the spinal cord; disturbances of this reflex process call forth ataxy. The diminution in the excitation which is conducted through the posterior roots causes a sinking in the excitability of the motor nerves, and thus calls forth irregular, flinging, excessive movements, because the patient applies too strong impulses of the will.

Benedict set up a theory of his own, on the basis of certain experiments of Harless, the correctness of which has with justice been challenged. Harless found, namely, that section of the posterior roots lowered the excitability of the mixed nerves belonging thereto, as he believes through the loss of a centripetal excitation, which is conducted through the posterior roots. According to Benedict, the ataxy now takes place through the failure of the regulating influence of the spinal cord, which is exercised upon the periphery through the medium of the posterior roots. He considers the ataxy as independent of the *perception* of peripheral sensitive excitation.

Great doubt is thrown upon all these views, first, by the opinion established by Goltz and others, that the centres for the co-ordination of movements, for the maintenance of the equilibrium and the locomotion of the body, do not lie in the spinal cord at all, but higher up. To be sure it may be conceded that the proof of this opinion, at least with regard to the human spinal cord, is not entirely conclusive; at the same time it is to a high degree probable that the actual processes of co-ordination, which regulate voluntary activity, do not take place in the spinal cord at all. If the spinal cord were really possessed of the co-ordinatory functions ascribed to it, then—as was correctly urged against the views of Jaccoud--a decapitated animal ought to be able to stand, or, having once been started in walking, ought to be able to walk further.

The experiments of Cyon, however, are out and out irreconcilable with the reflex theory, and thus this author has himself refuted his own views. He has shown that section of the posterior roots (which are supposed to have caused these reflex manifestations) causes no interference with ordinary movements (jumping, hopping, swimming, etc.), or at most interferes only with the most complicated movements, for the explanation of which the simultaneous disturbance of sensibility undoubtedly suffices. But we must certainly agree with the authors here cited in the opinion that *conscious* sensation, or the disturbance of its tracts, cannot possibly have anything to do with the ataxy. But the recently discovered fact of the apparently almost constant failure of the reflex action of the tendons in ataxy at least leaves the remote possibility open that disturbances of *certain* reflex tracts in the spinal cord, which act in an entirely unknown way in the co-ordination of movements, may have a relation to the ataxic disturbances of motion. This might be more carefully investigated in the future.

The view advanced by Leyden originally regarded the disturbances of *conscious* sensibility as the essential cause of the disturbance of co-ordination, and Leyden has taken much pains to demonstrate the disturbances of conscious sensibility in all patients, and asserts that these always exist to a degree about corresponding to the degree of ataxy. He therefore also attaches the greatest value to the careful examination of conscious sensibility. Almost all other authors have expressed themselves as opposed to this significance of conscious sensibility, and so of late Leyden himself also admits that it is *not conscious* sensibility that lies at the foundation of these co-ordinatory functions, but that probably these unconscious centripetal excitations within the spinal cord are conducted through the same tracts as the conscious sensations.

Leyden has most recently defended his theory over against the exceptions taken thereto, and expresses himself to the effect that, even though the question of the origin of ataxy through disease of the posterior columns be not satisfactorily resolved, *the cause of ataxy is to be sought in the loss of the connections leading in a centripetal direction between the periphery and the centre of co-ordination.*

We must admit that Leyden's view has something seductive about it, and that the way in which he has sought to refer the ataxic disturbance of locomotion back to a disturbance of the muscular sense appears to us quite plausible. We would also accept this theory without hesitation if a whole series of well-established facts did not exist which make this doctrine a logical impossibility. We have already adduced these facts at another point, and have drawn the conclusions resulting from them. We can furthermore refer to the new work of Friedreich, which brings forward such facts in still greater completeness, and radically refutes the deductions of Leyden. We may here perhaps add the cases of Trousseau and Bourdon, in which there was distinct ataxy with no anæsthesia of the skin or muscles; then a case of Ebstein's[1] in which there was sclerosis of the posterior columns accompanied by a high degree of ataxy without any disturbances of sensibility; finally a case of Engesser's,[2] from Kussmaul's clinic, in which muscular feeling was extinguished, cutaneous sensibility diminished, and Romberg's symptom present, *without* any trace of ataxy.

We must attach special value to the newer cases of Friedreich (which we were able, in part, to examine ourselves) in which exhaustive tests of sensibility, applied in all directions, showed no trace of disturbance of cutaneous or muscular sensibility, although a high degree of ataxy existed. When Leyden seeks to weaken the force of these cases by saying that they appear to him to deviate from the usual type of tabes, and are, therefore, "not directly to be credited to his theory," we can only reply that, from the standpoint of scientific logic, it appears to us, in such a dilemma, where well-established and undoubted *facts* will not fit a theory, much more correct to let the theory fall, than, for the sake of the theory, to reject the facts as inapplicable.

In like manner we cannot accept the rejection, proposed by Leyden, of the case of Spaeth and Schueppel (page 89). We do not see why "the turning to any theoretical account of so exceptional a case" should be "impossible." When such a case has been so accurately and repeatedly investigated, and that with special reference to Leyden's theory, it can surely be turned to account, even if it should throw the highest degree of doubt upon that theory. And when Leyden adduces, as the main argument against this case, that we might just as well conclude therefrom that disease of the posterior columns has nothing to do with ataxy, we must confess that this conclusion does not appear to us by any means so absurd, and that this very case of Spaeth and Schueppel furnishes one of the grounds on which we advocate the possibility, and perhaps probability, that the co-ordinatory tracts may *not* lie in the posterior columns themselves, but only in their neighborhood.

At all events, it would require *new*, well-confirmed, and unequivocal facts to make the theory of Leyden tenable; it can, in our opinion, simply *not* be maintained on the basis of the facts and observations now known. The disturbance of sensibility, as far as it has been possible thus far to investigate and confirm it, can-

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[1] Deutsches Archiv f. klin. Med. X. p. 595.

[2] Ibidem. XVII. p. 577.

[illegible] ataxy [illegible] may perhaps have some influ- [illegible] and certainly has none in [illegible] may indeed be [illegible] increased by the [illegible].

[illegible] reflex action and [illegible] of the vestibular may [illegible]. But I can [illegible] that the failure [illegible] in any constant normal [illegible] ataxy with [illegible] ataxy with fail- [illegible] symptom persisted [illegible].

[illegible] which make the ataxy de- [illegible].

[illegible] dealing with a disturbance of [illegible] as was taught by the theo- [illegible] deal with an [illegible] made to depend on [illegible] co-ordination lies there.

[illegible] a disturbance of sensibility [illegible] of an immediate disturb- [illegible] movements.

[illegible] of muscular anaes- [illegible] takes place by [illegible] spinal cord which he looks upon [illegible] all reflex theories.

[illegible] the disturbance of some definite [illegible] presides over the function of the co-or- [illegible] would seem to have to be sought in part in the [illegible] spinal cord [illegible] Topinard wrote at a time when [illegible] of the centres of co-ordination outside of the spinal cord was not yet [illegible].

[illegible] that ataxy is caused by a disturbance of [illegible] influence of the centre of co-ordination [illegible] outside of the cord, to the motor nerves. These tracts [illegible] posterior columns.

Our own view, already expressed above, is also to the effect that ataxy depends *on a disturbance of co-ordinatory* tracts lying within the spinal cord, and leading in a centrifugal direction.

We are therefore forced to the conclusion that there must be co-ordinating tracts in the spinal cord, tracts which maintain the communication between the centres of co-ordination and the peripheral motor nerves, and that ataxy can be explained only through a disturbance of these tracts. The question of *where these tracts lie* naturally obtrudes itself. Naturally, we first think of the posterior columns. But there are some objections to this view. One is, that cases of extensive degeneration of the posterior columns have been observed without any ataxy; another argument against it is the Spaeth and Schueppel case; another is perhaps the so frequent involvement of the gray matter and of the lateral columns in the later stages of tabes. We would therefore not regard the question as decided with entire certainty; we have already previously expressed ourselves on this question (page 95), and have scarcely anything to add to what was said there. Ought not, in the future, more attention, perhaps, to be given to the "direct tracts from the cerebellum to the lateral columns" which have been studied by Flechsig? At all events, many questions here remain to be solved by future investigation.

The fact of *swaying on closing the eyes*, previously referred to, has been regarded as having various different relations to ataxy, and as being a sign that ataxy was increased by closing the eyes. We have already previously shown that this is not correct. In fact, there are numerous instances where the swaying is present long before ataxy arises; and others in which, while the ataxy is great, closing the eyes causes no increase in the swaying; in these latter there are no disturbances of sensibility. Therefore this symptom, which, it is true, is seldom absent, remains chiefly as the sign of the existence of disturbances of sensibility, especially of the disturbance of muscular sense. If, nevertheless, in some patients suffering from tabes, there is a slight increase of ataxy on closing their eyes, this only proves that a moderating and directing influence can be exercised over the disturbance through the sense of sight, after the withdrawal of which the disturbance stands forth in its true magnitude.

Among the remaining motor disturbances the first that deserves notice is the *motor weakness*, which may afterwards increase to complete paralysis. As a rule, the "complete retention not have anything essential to do with the ataxy; it may perhaps have some influence in determining the *form* of the ataxic manifestations, but probably has none in producing them. The ataxic movements, in walking, for instance, may indeed be somewhat modified in their method of appearance, and perhaps increased, by the disturbance of sensibility, but they cannot be called forth by this alone.

It is possible that the testing of "unconscious" sensibility, of reflex action, and especially the more careful following up of the reflex action of the tendons, may afford some new fixed points for this theory. This remains to be seen. But I can already state, on the ground of some cases quite recently observed, that the failure of reflex action of the tendons probably also does not stand in any constant causal relation to ataxy. I have seen two cases in which there was undoubted ataxy with distinct increase in the reflex action of tendons, and one case of ataxy with failure of the reflex action of tendons (after diphtheria); this latter symptom persisted after the ataxy had long since disappeared.

For the present we consider those theories admissible which make the ataxy dependent on *a disturbance of the apparatuses of co-ordination themselves.*

It is out of the question to suppose that we are dealing with a disturbance of centres of co-ordination lying outside of the spinal cord, as was taught by the theories of Duchenne and Eisenmann. In tabes we have undoubtedly to deal with an affection of the *spinal cord;* the ataxy can, therefore, hardly be made to depend on a disturbance of the *cerebellum*—if, indeed, the centre of co-ordination lies there.

Finkelnburg considers the explanation of ataxy by a disturbance of sensibility as entirely untenable; according to him, it is a question of an immediate disturbance of the power properly to co-ordinate muscular movements.

Topinard likewise denies that motor weakness or cutaneous or muscular anæsthesia has any influence on ataxy; he considers the rather that this takes place by a disturbance of the co-ordinating function of the spinal cord, which he looks upon as a reflex power. His opinion is open to the same objection as all reflex theories.

Spaeth says: Ataxy can only be explained by the disturbance of some definite apparatus which, under normal conditions, presides over the function of the co-ordination of motion. This apparatus would seem to have to be sought in part in the gray substance of the spinal cord. Spaeth, like Topinard, wrote at a time when the situation of the centres of co-ordination outside of the spinal cord was not yet demonstrated.

Friedreich's opinion is to the effect that ataxy is caused by a disturbance of those tracts in the spinal cord which conduct the influence of the centre of co-ordination (which is to be sought outside of the cord) to the motor nerves. These tracts lie in the posterior columns.

Our own view, already expressed above, is also to the effect that ataxy depends on a disturbance of co-ordinatory tracts lying within the spinal cord, and leading in a centrifugal direction.

We are therefore forced to the conclusion that there must be co-ordinating tracts in the spinal cord, tracts which maintain the of motor weakness are present, there is some, even though but slight, involvement of the lateral columns or the gray substance. This, however, can only be decided by further investigations.

In tabes there is but very little to be seen in the way of *manifestations of motor irritation.* Here and there, particularly in the earlier stages of the disease, slight jerking contractions may be observed, spasms of single muscles, occasionally also fibrillar contractions. Not unfrequently there are also stronger twitchings of entire extremities, and the like; but these occur almost always only in connection with lancinating pains, and, therefore, are doubtless to be regarded as reflex.

There is an entire absence of only so-called *muscular tension* in all genuine and typical cases; the limbs are limp, easily movable, and do not offer the slightest resistance to passive movements.

On the other hand, in the later stages, muscular tension and contractures may also arise, with the true paralysis, and may finally reach so high a grade that the limbs remain immovable in the position of extension or flexion, as they do in the later stages of multiple sclerosis or of chronic myelitis.

The statements of authors differ much with regard to *the relations of electrical excitability* in tabes. This is sometimes said to be heightened, sometimes normal, sometimes lowered. The truth probably is, in other words, that the conditions of electrical excitability vary in different stages of the disease.

In my own examinations—and I pay no regard to any that are not made by my own method, which alone is applicable in such cases—I found the faradic and galvanic excitability, in a whole series of cases, to be quite normal in respect both to quality and to quantity.

In another series of cases I found a distinct, even though but slight *increase in the faradic and galvanic excitability* in the domain of the peroneal nerves, without any qualitative alterations.

And finally, in still another group of cases, I found a more or less distinct *diminution of electrical excitability* in the domain of the peroneal nerves, likewise without qualitative changes.

On the whole, I have received the impression that in the earlier stages of tabes there is rather an increase, in the later stages rather a diminution, of electrical excitability to be demonstrated. In one case I observed this directly, finding the electrical excitability at first materially increased, and afterwards diminished. Still, in view of the small amount of material before us, and the very variable intensity in the course of the disease, it is difficult to arrive at a definite conclusion with regard to these relations.

I have thus far not found any qualitative changes of the galvanic excitability in the domain of the peroneal (anomalies of the law of twitching [Zuckungsgesetz]); in particular have I never been able, in spite of careful search, to confirm what is claimed with regard to the existence of a greater tendency to twitchings at the opening of the circuit. Only in one case was I able to demonstrate, in the region of the ulnar, the occurrence of the AnSZ before the KaSZ, and a preponderance of the former over the latter, with low powers of the current.

Certainly, in the present situation of affairs, there is no great advantage to be gained, in the diagnosis or prognosis of tabes, from electrical examinations; we will therefore give no further time to the subject.

Disturbances of Reflex Action.

These have hitherto, it appears to us, received perhaps too little attention; in most histories of cases statements on this subject are very incomplete or are altogether wanting.

So far as the *reflex action of the skin* is concerned, it presents no sort of constant or essential anomalies. According to my own observations it appears to present no considerable deviations from the normal; that is to say, reflex action is generally present, more or less well marked, sometimes very active, sometimes hard to call forth. I have but very rarely found it absent; still it is known that this may be the case, even in health. In future these conditions should be more carefully observed, and it might perhaps be desirable in all cases more accurately to test the reflex action originating in the sole of the foot, that of the cremaster muscle originating from the inner surface of the thigh, and that originating from the skin of the abdomen.

Since writing the above I have carefully tested the reflex action of the skin, in this respect, in eight typical cases of tabes. In all of them the reflex action of the tendons was completely extinguished. Reflex action of the skin was retained in all of them, though in a very varying degree in different individuals. Sometimes the plantar reflex was very active, sometimes the cremaster and the abdominal reflexes were more marked, but in no case was there an absence of all the skin reflexes at once. No definite relation was found to exist between the reflex action of the skin and the degree of ataxy present, nor did the former bear any relation to the degree of disturbance of sensibility present. I have thus far merely received the impression that the reflex action of the skin behaves the same in persons with tabes as in those who are well, the latter also reacting with very variable degrees of intensity to cutaneous irritants capable of producing reflex action. Further investigations on this point are to be desired.

The condition of *reflex action of the tendons*, on the contrary, is very remarkable in tabes, and probably also very important. Westphal[1] first called attention to the entire absence of the reflex action of tendons in tabes. Since then I have always paid attention to this symptom, and can fully confirm Westphal's statements. In all typical and well-developed cases of tabes the reflex action of the tendons is entirely wanting, even though reflex action of the skin may be present and even increased. In only one case which I count as tabes, but in which there was, as yet, no ataxy, but only some initial symptoms, could the presence of reflex action of the tendons of the patella be demonstrated. Wherever the case had advanced to the development of ataxy, or even to slight indications of the same, reflex action of the tendons was entirely lost.

It would, of course, be in the highest degree interesting to know more accurately *when* reflex action of the tendons is extinguished—whether it disappears *before* or *after* the occurrence of ataxy, whether it disappears in the initial stage already, and thus to be able to establish the early diagnosis of tabes and the like. I have thus far labored in vain to clear up these questions. They require years of observation of single cases, or very favorable accidental circumstances. This matter is therefore commended to the study of our professional brethren.

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[1] Arch. f. Psych. u. Nervenkrankheiten. V. p. 819.

At all events, the great constancy of this symptom lends it a high diagnostic significance. It certainly stands in no demonstrable relation to cutaneous or muscular sensibility, nor to the reflex action of the skin. I have seen the reflex action of tendons fail when the cutaneous and muscular sensibility were entirely intact and when they were greatly diminished, when the reflex action of the skin was normal, raised, or lowered. This symptom, therefore, seems to be entitled to a certain isolation in the symptom-picture of tabes. At the same time there are complicated cases—and we shall return to this again under the head of Spasmodic Spinal Paralysis—in which, together with ataxy and other symptoms of tabes, an increase in the reflex action of tendons shows itself, together with other manifestations of spasmodic spinal paralysis. These are certainly not typical cases of tabes, and they constitute well-characterized exceptions.

Disturbances of the Organs of Special Sense.

We would mention, first, the *paralyses of the muscles of the eye*, which play a not unimportant rôle in the clinical picture of tabes.

They most frequently affect the motor oculi and abducens, far less frequently the trochlear nerve, and manifest themselves by the corresponding disturbances of binocular vision, diplopia, strabismus, dizziness, etc. von Graefe calls attention to the fact that tabes patients who are suffering from diplopia show a strikingly small tendency to the blending of images in binocular vision, which is supposed to point towards a central origin of the affection.

These paralyses may arise quite transitorily, lasting, with varying intensity, for a couple of days, weeks, or months, then disappearing, and returning again after a longer or shorter time; in the later stages of the disease, however, permanent paralyses of the muscles of the eye doubtless also occur.

Not infrequently *disturbances of accommodation* also take place, pareses of the same, and *alterations in the pupils*. The latter are sometimes contracted or dilated on one side; but more frequently there is a bilateral, uniform, generally pretty considerable contraction of the pupils, which presents the characters of spinal myosis; that is, the pupils do *not* react to the stimulus of light, but do still react distinctly to accommodative impulses (Arg. Robertson, Knapp, Leber, Hempel). This condition is generally permanent.

The occurrence of all these changes in the muscles of the eye is by no means rare in tabes. If we embrace them all in our estimate, including the slight and transitory disturbances, the differences in the pupils, the spinal myosis, then we shall be able to establish the existence of such changes in more than one-half of all cases. The more prolonged and permanent disturbances, on the contrary, may probably not arise in more than one-third to one-fifth of all cases.

The transitory disturbances occur with special frequency in the initial stage of tabes, and are to some degree characteristic of the same, although by no means as constant as Duchenne at first stated. But paresis of the ocular muscles may arise in every stage of tabes, and it is just the severer forms of the same which generally appear only in the well-pronounced disease.

In a pathogenetic point of view, the most important item, as influencing these changes, is doubtless the disease of those fibres of the ocular nerves which run together in the medulla oblongata. Probably slight diseases of the nerve-tracts here situated, and of their nuclei, are to be looked upon as the causes of paresis. Still, such diseases have not hitherto been anatomically demonstrated; neither has any direct connection with the degeneration of the posterior columns been thus far established, either anatomically or physiologically. Pierret declares these paretic disturbances of the muscles of the eye (as well as of the muscles of the face and of mastication, which sometimes occur) to be dependent on primary irritation or disturbance in the tract of the trigeminus, which participates in the disease in a manner quite analogous to that of the posterior spinal nerve-roots, and he regards them, therefore, as paralyses of sensitive origin (reflex paralysis, etc.). The idea that, particularly in prodromal diplopia, it is a question of rheumatic paralysis of the muscles of the eye, consequent on taking cold, will find but little support. The changes in the pupils would, first of all, suggest the involvement of the cilio-spinal centre and the oculo-pupillar tracts emanating therefrom and lying within the cervical portion of the cord ; but here, too, we are thus far without the support of any anatomical proof.

The *nystagmus* observed by Friedreich in a series of cases, and more accurately described by him, is a peculiar, but, on the whole, a very rare disturbance of the muscles of the eye. This is always a bilateral nystagmus, which follows partly a horizontal, partly a vertical, or probably also a diagonal direction, which does *not* occur during rest, but does occur at every attempt to fix the eye. If an object is moved in various directions in the field of vision, irregular, jerky twitching of the ball of the eye takes place, being all the more prominent the more the object is approximated to the outer limits of the field of vision. In the higher grades of the trouble, nystagmus also occurs when the eyes are fixed upon an object held at rest, but even then it is absent when the glance is quiet and not fixed. The movements are slower and less regular than in the ordinary nystagmus, dependent only upon disease of the eye, and are principally to be distinguished from the latter by the fact that they only arise on voluntarily moving the eye or fixing the glance. Friedreich is undoubtedly right in considering this nystagmus as a form of ataxy of the movements of the eye, and in setting it up as ataxic nystagmus, over against the ordinary form of the disease.

Friedreich seeks the cause of this nystagmus in a disturbance of the co-ordinatory tracts which lead from the centres of co-ordination to the nuclei of the nerves of the ocular muscles lying on the floor of the fourth ventricle, and declares himself to be of the opinion that nystagmus does not arise in spinal disease until the medulla oblongata is involved. This proposition also still lacks anatomical proof in tabes. Pierret explains these phenomena also on the ground of the primary disease of the sensitive root-tracts of the trigeminus in the medulla oblongata.

The occurrence of ataxic nystagmus is rare in tabes, and has thus far only been observed by Friedreich in five of the nine peculiar cases described by him. It belongs to the later symptoms, and becomes associated with the other ataxic manifestations only after the disease has existed for a number of years (five to twenty-one).

But the *atrophy of the optic nerve*, which is unfortunately very frequent in tabes, is of much more importance than the disturbances of the visual apparatus thus far considered, and is much harder for the patient to endure. It belongs to the saddest complications of this already melancholy disease; the helplessness caused by the ataxy is aggravated beyond all measure by the blindness added thereto, and the sources from which the patients can obtain comfort and oblivion amid their suffering are materially diminished.

The trouble begins with slowly or rapidly advancing *diminution in the sharpness of vision*, which soon increases to *amblyopia*, and finally to *amaurosis*. The *field of vision*, which is at first slightly veiled and cloudy, *becomes increasingly narrowed*, generally from without inward, sometimes from without and above, sometimes more from below. Examination shows that this narrowing of the field of vision does not take place in a uniform manner, but with entering angles. Finally, the blind area involves all but a spot towards the inside, which allows the patient but a limited and insufficient amount of vision.

Color blindness can usually be demonstrated in patients before any limitation in the field of vision. Generally the perception of green is lost first, then that of red, finally that of yellow and blue; still, deviations from this order are not excluded.

In such cases the pupils are frequently contracted rather than dilated; their reaction to light entirely suspended. In the earlier stage many patients show an increased sensitiveness to bright light, and therefore see somewhat better in the twilight than in bright daylight.

Ophthalmoscopic examination reveals the signs of so-called *white atrophy of the optic nerve.* At first there is a slight grayish discoloration of the papilla, which gradually grows paler and finally appears quite white and sharply outlined. The arteries at the same time show a progressive narrowing, but otherwise the retina remains quite unaltered. These changes are easily to be distinguished from the ophthalmoscopic picture of neuritis optica and congested papilla, and according to French authors they are so characteristic that the existence or the approach of tabes can be recognized from them alone.

Atrophy of the optic nerve in tabes is always—with quite rare exceptions—a decidedly progressive malady, and leads without interruption to complete amaurosis. In different cases, it is true, this occurs with very variable rapidity; sometimes it requires only weeks, sometimes months, or even years, for its accomplishment. Occasionally the malady comes to a stand-still, even after it has lasted for a comparatively long time. Sometimes the trouble is limited for a considerable time to one eye, but it is far more common for both to be attacked and to become blind simultaneously or within a short time. Modifications between these two extremes are of course manifold.

Atrophy of the optic nerve belongs to the more frequent of the complications of tabes, but we have no accurate statistics as to its frequency. The statements of ophthalmologists are perhaps generally too high. I myself have seen it but eight times among about seventy cases. Topinard, on the contrary, gives "disturbances of vision" in 49 cases out of 102. Cyon, in 203 cases, finds 60 of amblyopia and amaurosis. More accurate statistics on this subject are to be desired.

Very commonly atrophy of the optic nerve arises in the initial stage of tabes; it may even be the first manifestation of the disease, and may, for a longer or shorter period, precede the lancinating pains and the other symptoms. The amaurosis caused thereby may even exist for years (up to ten years, Charcot) before other symptoms of tabes are added thereto.

The immediate cause of the amaurosis of tabes is, of course, the gray degeneration of the optic nerves, a typical gray degeneration entirely analogous to the changes in the spinal cord, and one which we have learned to know more accurately through the labors of Leber. The disease begins at the periphery of the trunk of the optic nerve and attacks the most central fibres of the same last. It always begins on the trunk of the optic nerve itself, in the divisions nearest to the eye, and spreads from here towards the centre to the optic tract, in rare cases also to the tubercula quadrigemina. The narrowing of the field of vision and the method of its development would alone enable us to decide with certainty that the change always begins in the trunk of the nerve and not in the tractus opticus (Foerster.)

What connection is there between this degeneration of the optic nerve and the sclerosis of the posterior columns? Probably there is no direct connection, for no anatomical continuity can in any way be demonstrated between the process in the optic nerve and that in the posterior columns. The idea which naturally suggests itself, that the disease in the posterior columns of the cord might interfere with trophic influences exerted on the optic nerves and thus cause their gray degeneration, can also hardly be maintained in the face of the fact that atrophy of the optic nerve often for a long time precedes all manifestations of the spinal disease. Consequently for the present there is not much of anything better left for us than the conclusion that gray degeneration "is developed simultaneously or successively at various points in the central nervous system especially predisposed thereto" (Foerster.)

Disturbances of hearing very rarely arise in tabes. Imperfect hearing has indeed been observed here and there (Leyden, Remak, Vossius), but in the majority of instances it was doubtless due to an accidental concurrence of two diseases, each frequent enough in itself. This accidental character of the deafness in tabes has been especially demonstrated by Lucae[1] in two cases. But it seems probable, from a case observed by me, that tabes may also be accompanied by purely or principally nervous deafness, which probably depends on atrophy of the auditory nerve and is thus analogous to atrophy of the optic.

The case was that of a Russian naval officer, who for some years had suffered from the manifestations of a somewhat complicated tabes. During the initial stage of the same, besides psychical disturbances, occasional headaches and *deafness* had arisen, the latter gradually increasing. When I first saw him the patient was suffering with moderate ataxy of the legs, with swaying on closing his eyes, was very easily fatigued, had a sense of numbness in his feet, slight weakness of the bladder, etc. My colleague, Prof. Moos, had the goodness to examine the organ of hearing more accurately, and to communicate to me the following results: July 21st, 1873. "On both sides hyperæmia of the handle of the malleus, and slight cloudiness of the mucous membrane. *Right ear:* the tuning-fork could not be heard, either in the air or by conduction through the bones; hearing of speech, null. A watch of thirty feet hearing distance heard only on pressure. *Left ear: C'* and *C''* [tuning-forks] not heard at all through the cranial bones, and but feebly through the air; from *a* upward the vibrations are heard both through the cranial bones and by way of the air. High tones can be heard through the air at a distance of seven paces. With the watch, H. D. = two inches; conduction through cranial bones very good; spoken words understood at a distance of four feet." There was, therefore, probably atrophy of certain fibres of the auditory nerve. On the 7th of August, 1873, Prof. Moos reports: "*Right ear: C'* not heard; *C''* heard; *a* also heard through the air; no conduction through cranial bones; watch heard a distance of two inches; words distinguished at a distance of one foot. *Left ear:* tuning-forks of a low pitch are now heard through the cranial bones, as well as through the air; high tones are heard through the air at a distance of ten or twelve paces; watch, eight inches; spoken words, six paces."

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[1] *Lucae*, Ueber Schwerhörigkeit bei grauer Degeneration des R.-M. Verh. d. Berl. med. Gesellsch. Bd. I. S. 127. 1866.

Appreciable improvement had therefore followed the treatment employed (galvanism).

But, at all events, the question of the occurrence of affections of the auditory nerve in tabes requires more accurate investigation.

Disturbances of taste and smell have indeed been observed here and there in tabes, sometimes with simultaneous anæsthesia of the mucous membrane of the tongue and mouth; they are, however, of very subordinate significance.

Disturbances of the Action of the Brain.

These are comparatively rare in tabes. In the typical form of the disease, intelligence, memory, and disposition are likely to remain entirely intact. The state of mind of the patients is often even remarkably cheerful and contented, and they often endure their serious troubles with wonderful good humor. At other times, it is true, one also finds these patients depressed, unhappy, very irritable and moody, tormented with sleeplessness, quarrelling with their fate.

More serious *psychical disturbances*, through complication of the tabes with an affection of the brain, are but seldom observed. These may precede the tabes for many years, or may become associated with it after it has existed for years, or, finally, may not arise until the terminal stage.

Since the admirable labors of Westphal special attention has been directed to the connection between tabes (that is, gray degeneration of the posterior columns) and *the progressive paralysis of the insane.* We can here only briefly touch on this interesting and far-reaching subject, and must refer our readers, for further information, to the original works on this subject, especially those of Westphal. According to this author, all, or at least the majority of the patients who present the picture of ordinary progressive paralysis, and, in so far as they present motor disturbances of the extremities, seem to have an anatomically demonstrable disease of the spinal cord (chiefly degeneration of the posterior columns). In these cases, therefore, the tabes is one of the manifestations of a more or less diffuse process of disease, extending over a great part of the central nervous system ; the tabes, at its very beginning, is combined with a psychopathic group of symptoms.

In another set of cases, however, the paralytic insanity is added to the spinal affection of tabes only after the latter has existed for years. Naturally, in all such cases, the importance of the spinal becomes subordinate to that of the cerebral disease.

The frequency with which the true *cranial nerves* are involved in tabes is very variable. We have already seen that the motor nerves of the eye and the optic nerves themselves are very often diseased. On the other hand, the involvement of the remaining cranial nerves not yet mentioned is among the greatest rarities in tabes. The trigeminus shows signs of irritation here and there (pain, paræsthesia, etc.), or it may be paretic conditions (anæsthesia, sense of numbness, disturbance of taste, etc.) which have been thoroughly established by Pierret, and referred to sclerosis in the region of the lower nucleus of the trigeminus in the medulla oblongata. Pierret attributes very great importance to this disturbance in the development of various "cephalic" symptoms of tabes. The *facial* nerve is but very rarely found implicated, and then it is principally the branches serving for the mimetic movements of the mouth. The *hypoglossal* has occasionally

been found affected (Friedreich). The
accessory, as well as the *glossophar*
cated comparatively seldom; difficul
tabes; palpitation of the heart, att
gastralgic troubles, and the like, do, i
certainly decided whether they are ca
nerves above named.

Disturbances of the Functions of

These are among the most co
Sometimes evidences of irritation a
parts; spasmodic dysuria, hyperæs
bladder, neuralgic (lancinating) pain
in the perineum or the neck of the bl
tion, or on defecating, have repeatedl

But *signs of paresis* are far more
part of the bladder, more rarely on th
various disturbances in the evacua
which we have previously depicted
endeavored to refer to their physiolo
larly the lighter difficulties with wh
flicted; severe and complete paraly
occur at all, or only in the latest stag

Patients either complain of slight
the bladder takes a longer time, an
stronger pressure and with some sub
is a moderate degree of incontinence,
desire to urinate, they must do so a
of the bladder occur more frequently
into the clothes or bed. It is but rare
to have recourse to the catheter or to
constant involuntary flow of urine an

After a longer or shorter time e
almost always show themselves, bu
stricted within moderate limits, and
interference.

For an explanation of all these di

discussion of the subject at a previous place; still, we must not fail to call attention to the fact that the sclerosis of the *posterior columns* in itself probably does not afford sufficient ground for these disturbances, but that, in order to explain them, we must probably assume a simultaneous disease of the gray substance. But these are all matters calling for still further examination.

The *disturbance of the functions of the rectum* is also likely to be but insignificant in tabes. The most frequent is anæsthesia of the anus, whereby patients lose the feeling of approaching or actual evacuations of the bowels, so that they not infrequently dirty themselves. True paralysis of the sphincter probably occurs only in the latest stages of the disease.

Disturbances of the Sexual Functions.

These are scarcely ever absent in tabes. *Weakness* is the most frequent and common evidence of this disturbance; still, evidences of abnormal irritation are not always wanting. At the beginning of the disease, in particular, these evidences of increased excitement are not rare. Trousseau and Hammond have observed increased sexual power at the beginning of the disease; Charcot, in one case, saw satyriasis. Not infrequently patients are possessed of a high degree of sexual excitability, so that their passions are at once aroused by the sight or the innocent touch of a woman. But this increased excitability is almost always already associated with signs of sexual weakness; if patients are still able to perform the act of coition, they still have premature ejaculation; discomfort, a sense of weakness and pain follow the act; such patients suffer from frequent pollutions, and cases are not infrequent in which a lascivious thought or word, or the mere sight of a woman, is enough to excite an escape of semen, which then takes place without an erection and without any libidinous sensation.

More frequently, however, there is *impotence*, beginning early and increasing rapidly. At first such patients can still perform the act of coition, but more rarely, with less power and pleasure. It is very common for patients with tabes to beget children even after the disease has lasted for several years. But gradually this power is more and more extinguished, erections become rarer and weaker, and finally fail altogether. As a rule, sexual desire is at the same time more or less completely extinguished; still, there are cases in which frequent pollutions and increased sexual excitability exist together with complete impotence.

It must finally be mentioned that some tabes patients may also retain perfectly undiminished sexual power for a very long time.

We have already previously intimated (p. 137) how imperfect our knowledge is as yet with regard to the more exact localization of these disturbances within the spinal cord.

In women with tabes, disturbances of the sexual functions are, as a rule, not observed. Menstruation, pregnancy, and the puerperal condition may run their course quite normally.

Vaso-motor Disturbances.

These fall very much into the background in tabes, and are but little studied. In many cases cold feet are very common; the occurrence of blue spots on the skin, increased or diminished secretion of sweat, especially suppression and disappearance of sweating of the feet; sometimes also a great tendency to the occurrence of "goose-flesh" (*cutis anserina*). The more accurate relation of all these disturbances to sclerosis of the posterior columns is not yet determined.

Trophic Disturbances.

These likewise belong to the infrequent symptoms; in fact, it is quite striking how admirable the general nutrition—the nutrition of the muscles and the skin—is in many tabes patients. It is not until the final stages of the disease that trophic disturbances are frequent, and only in single cases that they occur in the earlier and even in the initial stage.

On the skin, eruptions of herpes, of lichen, and of something like pemphigus are to be observed here and there. Bed-sores belong only in the last stage to the ordinary and almost constant manifestations, and then they present all the characteristics of chronic bed-sores, as previously depicted (p. 122), and are amenable to the same attempts at an explanation.

In the ordinary and typical cases the nutrition of the muscles remains for a long time entirely intact; there are tabes patients enough with splendidly developed, hard, and powerful muscles. Not until the latest stages, when there is already complete paralysis, do we often see a high grade of muscular atrophy set in, which, however, only represents simple emaciation of the muscles, and is not to be regarded as degenerative atrophy.

In occasional, rare cases, however, this complication appears earlier, with a high degree of atrophy of single muscles or of groups of muscles (atrophy of the ball of the thumb, hemi-atrophy of the tongue, atrophy of the calves of the legs or the thighs, etc.). These atrophies are doubtless, in the majority of cases, to be regarded as degenerative atrophy; they cannot possibly depend upon the sclerosis of the posterior columns, and their appearance points with all certainty to the fact that the pathological process in the spinal cord has extended further, to motor and trophic tracts. It is in the highest degree probable (Charcot, Voisin) that in such cases there is gray degeneration of the corresponding portions of the anterior horns of gray matter. The few cases of this kind which have thus far been more carefully examined with the naked eye argue in favor of this.

Those *disturbances of nutrition of the joints*—"arthropathies des ataxiques"—of which we have also previously spoken (p. 126), and which occur quite often in tabes, certainly belong to the most remarkable manifestations met with.

This joint affection generally develops itself in the initial stage, even before the ataxy is manifest. If any joint trouble appears later, it is usually in the upper extremities, when the disease spreads to them; therefore when the initial stage has been reached for these extremities. The joint most frequently attacked is the knee-joint; descending in the order of frequency, after that are the shoulder, the elbow, the hip-joint, and the wrist.

In describing this very characteristic affection we can be brief.

The scene opens with a sudden and high degree of swelling of the joint, caused by an accumulation of fluid, without pain, without redness and heat, and generally also without fever; associated with this there is a great and often widespread doughy swelling of the surrounding parts, so that the whole extremity may be extensively swollen; what is particularly striking is the non-sensitiveness even of joints that are greatly altered on active or passive motion.

If the joint affection is of a benign form all these manifestations may disappear again in the course of a few weeks.

Generally, however, the affection is of a character very disastrous to the joint; it results in progressive destruction of the articular extremities, sub-luxations or spontaneous luxations of the joints, causing notable deformities; thus at the knee-joint, in particular, an exceedingly striking hyper-extension of the leg is produced.

As a rule, it is easy to distinguish this joint affection of tabes from rheumatic inflammation of the joints or from arthritis sicca.

Probably the *abnormal friability of the bones* now and then observed belongs in the same category of disturbances. In one case Charcot saw numerous spontaneous fractures arise, and Blum found these to be occasioned by a rarefying ostitis.

It is in the highest degree probable that these trophic disturbances in the joints and bones are of neurotic origin; still we have as yet no *positive* demonstration of their dependence on disease of definite portions of the spinal cord. Probably disease of the gray matter of the anterior horns is to be held responsible for it. Charcot and Joffroy at least found disease thereof in a case of arthropathy; and in A. Pick[1] I find the notice of an observation of Westphal's, in which, together with alterations of the joints, atrophy of the large ganglion-cells of the anterior horns was found. In another case Charcot, on the contrary, found no changes whatever in the gray substance, though there was swelling of the spinal ganglia, which may, therefore, also possibly have an influence on these processes.

We must finally notice still another series of *rare symptoms* of tabes which we enumerate more for the sake of completeness than because they contribute particularly to the characterization of the disease.

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[1] Arch. f. Psych. u. Nervenkrankh. VI. p. 695. 1876.

First of all an *increased frequency of the pulse* may be mentioned, which is considered by some authors as a tolerably constant manifestation in tabes; the pulse is said to beat over a hundred and even up to a hundred and fifty to the minute. I have no personal experience in this.

Friedreich, in one of his cases, observed *profuse sweating, diabetes insipidus* and *salivation.* Bouchard claims once to have found acid saliva.

More importance attaches to the *gastralgic attacks*, the so-called "crises gastriques," which are by no means rarely to be observed, and the existence of which was first thoroughly established by Delamarre and Charcot. These are extremely severe cardialgic pains, arising in paroxysms, associated with nausea, retching, vomiting, dizziness, sometimes with diarrhœa, but generally with constipation, etc. In the severest forms there is swelling of the abdomen, rigors amounting to convulsive trembling, vomiting of watery mucus with bile and blood, very frequent heart's action, etc. These attacks may last one day or several, and may return every couple of weeks, thus greatly reducing the patient. They may arise at any period of the disease, in the initial or the ataxic stage, and evidently bear the greatest analogy to the severe attacks of lancinating pains. They may, doubtless, also be referred to the same anatomical causes as the latter.

The "crises néphrétiques" recently described by Raynaud, which present the most exquisite picture of severe renal colic and may cause the patient fearful torture, evidently show great analogy to the above. They are to be distinguished from true renal colic by the longer duration and more frequent return of the attacks, and especially by the absence of all disturbances in the urine (admixture of blood, gravel, renal calculi, etc.). The "rectal and urethral colics," described repeatedly, doubtless also belong in the same category of disturbances.

The "bronchial crises" first described by Féréol, attacks of spasmodic cough, difficulty of breathing and of swallowing, are

at least doubtful, as yet, so far as the
concerned (Bracht). Martin has rece
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were observed. In a case of tabes i
of suffocative cough (arising spontan
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Individual Clinical Fo

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The more abundant the amount
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nerve-pathologist of the present day
everything as tabes which was so re
We have, through many conflicts an

tained a keener eye for the symptom-picture of tabes; we are able, with greater certainty, to distinguish between the clean typical cases and the irregular, the complicated, mixed, and transition forms. We have progressed far enough to obtain a quite definite, sharply-outlined idea of tabes, so to speak, an ideal picture of the same, which we regard as the regular and normal one, even though it may arise in numerous varieties, the connection of which with the typical form is, however, easily recognizable. Any considerable deviation from this type always at once indicates an unusual manifestation of the disease, a complication of the same, a combination with other allied forms of disease. And these deviations occur but comparatively seldom.

Regarded from this point of view the sifting of the numerous individual cases of tabes appears tolerably simple. As far as we can see, there is, first of all, a large group, comprising by far the greater number of cases of tabes, and which we may, therefore, regard as the regular and typical form of the same. This has been depicted in the preceding pages.

Next to this comes a far smaller group, containing fewer cases, but likewise well characterized and distinctly deviating from the typical form. This group comprises the cases of "hereditary ataxy" described by Friedreich, which agree among themselves in a remarkable manner.

All other forms may probably be best regarded as merely varieties of these two types; as cases in which single symptoms attain peculiarly prominent significance, or new and unusual symptoms appear, or the signs of other diseases of the spinal cord (or probably also of diseases of the brain) are mixed with the symptoms of tabes.

We may here be permitted a brief characterization of these different forms.

The regular, typical form of tabes.—Special regard has been had to this in the preceding pages, and it therefore only requires to be sketched in very brief outlines.

The beginning of the disease, which usually attacks *men during middle age*, is with *lancinating pains;* with *disturbances in the muscles of the eyes* and the *optic nerves;* with disturbances in the sensibility and motion of the lower, much more rarely of the upper extremities, such as *paræsthesias*, the *sense of a tight girdle*, *slight anæsthesias*, *swaying on closing the eyes*, a great *feeling of fatigue*, and the fact of getting tired soon, *lowering of the motor power and endurance;* and generally also *weakness of the bladder and sexual weakness.*

During a second stage of the disease, while the symptoms of the first stage gradually increase, disturbance of the co-ordination of motion—*ataxy*—is added. This spreads slowly from above downward. Patients grow increasingly helpless; there are *well-marked disturbances of sensibility*, partial paralysis of sensation, retardation of the sense of pain, though rarely complete anæsthesia. *Reflex action of the tendons extinguished. Vesical and sexual weakness increased. Motor weakness* more distinct. General nutrition still good; psychical functions remain intact.

Finally, in the last stage, there is a transition to *true paralysis*, paraplegia, contractures, muscular atrophy, bed-sores, *progressive cachexia*, which finally induces death with or without intercurrent diseases.

Naturally there are numerous deviations from this general picture of disease (although a strikingly large number of cases show a uniformity extending even to the smallest minutiæ). Of course, in many individual instances, the picture of the disease will assume a somewhat different form, according as the one or the other symptom predominates, or arises earlier or later, according to the slower or more rapid progress of the disease from the lower to the upper extremities, according to the complications or rare symptoms that arise earlier or later. But the typical picture of the disease will still always be recognizable in its most essential features.

But in our opinion it has not yet been possible, on the ground of these differences in the symptom-picture of tabes, to establish different forms of the same, among which all the individual cases could be classified. Nor is it as yet possible, on the ground of the symptom-picture, to determine whether we are dealing with an inflammatory or a simply degenerative form of sclerosis of the posterior columns, nor to classify the cases according to their anatomical seat at different heights within the spinal cord. To be sure, with our present knowledge, it will be easy to distinguish in each individual case about how high the anatomical process extends in the spinal cord, whether the lumbar portion alone is involved, or the dorsal and cervical portions as well; furthermore, whether the cranial nerves are implicated, whether the bulb of the medulla oblongata is likewise affected, etc. After our repeated explanation of these points no one need remain in doubt about them.

On the other hand, one may be justified in doubting whether it is a possible thing to determine the seat of the disease, not only by the localization, but also by the *quality* of the symptoms, and whether one is justified in accepting this accidental seat of the disease as a sufficient ground for distinguishing different forms of the same.

Remak, in this way, distinguished not less than six forms of tabes, all of which he thought he could recognize by certain symptoms: a tabes lumbalis, lumbo-dorsalis, dorsalis ascendens, cervicalis, basalis, and cerebellaris. Cyon has tried to simplify this division so far as to accept only three forms, which he characterizes as follows: 1. *Basal form*—Begins with disturbances in the muscles of the eye; progressive atrophy of the optic nerve; pupils not contracted; ataxy always present; anæsthesia frequent, especially in the upper extremities; psychical alterations may arise. 2. *Cervical form*—Contracted pupils; severe, boring pains in the extremities; ataxy rare; the eye affected only later, never any paralysis of the muscles of the eye; always irritability of the genitals, and impotence. 3. *Dorsal form*—Anæsthesia rarely absent; bladder trouble and difficult defecation frequent; pains not as severe as in the first two forms; paræsthesia and the sense of a tight girdle common; never any eye trouble, only sometimes dilatation of the pupils.

This indefinite characterization of itself shows that all possible transitions of the individual forms amongst themselves must take place, and if one takes the trouble to test a large number of cases with reference to their belonging to one of the three groups, he will find that but very few of them fit into the corresponding mould, but that most of them directly represent transitions between the different forms. We therefore regard it as a hitherto unsatisfactory task to attempt such a division; it will suffice to content ourselves with recognizing tabes as such, and, in certain cases, also with determining approximatively its more exact seat at different heights within the spinal cord.

The Friedreich form of tabes.—The disease is developed *on the basis of a hereditary or family predisposition.* The cases thus far known are distributed among three families, in each of which several brothers and sisters were attacked, *especially the female members* of the families. It begins at a very early period of life—between the twelfth and eighteenth years—perhaps in connection with the development of puberty (or with a congenital imperfect development of the spinal cord ?). Lancinating pains are very rare at the beginning; motor disturbances, on the contrary, occur very early, quite at the beginning, in the form of *well-marked ataxy*, which spreads rapidly to the upper extremities, even arising simultaneously in the lower and upper extremities. *Co-ordinatory disturbance of speech; ataxic nystagmus. No disturbances of sensibility, or but very late and insignificant ones;* the sensibility of the skin and muscles remains quite intact for many years. No swaying on closure of the eyes. *Reflex action of the tendons extinguished.* No disturbance of the bladder nor bed-sores. No psychical disturbance, no tremor, no amaurosis. Paresis and contractures, atrophy of the muscles, weakness of the bladder, appear only in the latest stages. *Remarkably long duration of the affection* (reaching to over thirty-two years).

The cases recently described by Kellogg—unfortunately in an extremely imperfect manner—probably belong here. The disease was developed in two brothers, in each at the age of six years; there were several similar cases in other branches of the same family.

In addition to these well-marked types of the disease there are numerous cases, in practice, which deviate more or less from the type without having to be thrown out of the general group of tabes. We have already above called attention to the fact that the different forms of chronic myelitis, in which we also include tabes, are by no means always sharply separable, but that they not infrequently extend beyond a certain definite domain in the transverse section of the cord, and even beyond the cord itself, and thus produce combined and complicated symptom-pictures, the true significance of which can only be attained through an accurate knowledge of the typical forms. These remarks also hold good, to the fullest degree, for tabes; here, too, there are numerous complicated and transition forms, which, however, do not possess regularity enough to justify their being set up as a well-characterized form of the disease. A comprehensive and expert judgment will very often be able to recognize the typical germ in the abnormal symptom-picture, and will correctly grasp the significance and the pathogenetic basis of each unusual and complicating symptom. These remarks may suffice to call attention to these irregular forms, to only a few of which we can cursorily allude. Sometimes it will be simply impossible to establish a clear-cut diagnosis of any customary form of the disease, but we shall have to content ourselves with establishing the existence of a more or less irregularly localized and extended chronic myelitis.

In a certain number of cases manifestations of *motor weakness and paresis* come into prominence very early, while the ataxy is less pronounced, but the other symptoms are as usual; in this way symptom-pictures may be reached which suggest paraplegia —emaciation and atrophy of the legs may become associated with it. In such cases one doubtless has a right to suppose an early involvement of the lateral columns and the anterior gray substance, both of which conditions, indeed, have been anatomically demonstrated repeatedly.

In other cases the *lancinating pains* stand forth with striking prominence in the picture of the disease; they are for a long time the only and afterwards certainly the most tormenting manifestation, and arise during the whole course of the disease, sometimes in rarer, sometimes in more frequent paroxysms, which, by their intensity and duration and the other symptoms associated with them, may become an excessive torment to the patients. The attempt has been made to organize these cases into a separate form, "tabes dolorosa," and Remak especially claims this character for his "cervical form" of tabes. We have not yet been able to satisfy ourselves that this is justifiable; we have observed such severe and repeated attacks of pain occasionally in most of the forms of tabes.

In other instances, again, manifestations of *pain in the back, spinal sensitiveness, circumscribed or diffuse cutaneous hyperæsthesia,* and the like, are extremely prominent, and in such cases one is doubtless justified in thinking of a meningitic complication of tabes. The relations of sclerosis of the posterior columns to the accompanying spinal meningitis may be very varied, and thus a great variability may appear in the individual cases belonging under this head.

Finally, it is not rare for tabes to be complicated with *psychical disturbances*, as has already previously been intimated; here, too, the most varied combinations may arise. We may not make special forms of tabes out of these, but must simply conclude that the occurrence of the psychical disturbance signifies nothing more than a disturbance in the brain, which is added to the disease in the spinal cord.

We might give many more similar hints with regard to irregular forms of tabes, but it would be superfluous. We only wish once more to emphasize the fact that these irregular, complicated cases of tabes constitute decidedly the minority over against the regular, typical forms of the disease.

Course—Duration—Terminations.

With regard to the method of development and course of tabes, we have but little to add to what appears from the general description of the disease.

Tabes is a disease of a remarkably chronic course, and it seems to us extremely questionable whether what has of late repeatedly been described as "acute ataxy" has any relation whatever to tabes and to sclerosis of the posterior columns.

The development of the trouble is accordingly slow, chronic, extending over months, not rarely over a whole series of years. Only in rare cases has the disease hitherto been seen to develop itself so far within a few weeks or months as that a positive diagnosis could be made. Generally, however, we observe the slow development described above, the rise of single symptoms which remain isolated for a long time, to which others are gradually associated, until in the course of months and years the picture of the disease is complete. This development usually begins in the lower extremities; still, single cases have also been observed in which the upper extremities were first and prominently attacked with the symptoms of tabes (Vossius). In not altogether rare instances a unilateral development may also be observed, and the existence for a longer period of exclusively or mainly unilateral manifestations.

When the disease has once become developed, its further course usually leads slowly from bad to worse. More or less rapidly the intensity of the individual symptoms increases, new symptoms arise, and the condition of the patient grows ever more deplorable.

This does not exclude the possibility of more or less considerable fluctuations: slight or even considerable improvement may go on for months and years, then again to give place to an unfavorable change; during the summer patients feel better and more comfortable, while the winter always makes them worse again. Generally, however, the evil influence of winter outweighs the good of summer; it is but very rarely that the tendency to improve gains the upper hand, and the patients then advance gradually towards recovery.

A prolonged arrest of the disease is quite common in tabes; when it has reached a certain point of development, when it has, perhaps, progressed so far as to compel the patient to use a rolling-chair, he will be seen remaining in about the same condition for years, and even for decades, dragging out a miserable existence, often with tolerable cheerfulness and resignation.

These arrests may then occasionally be interrupted by external influences and injuries (by taking cold, by an emotional disturbance, a disastrous attempt at a bath-cure, mistaken therapeutic measures, or the like), and the occasion be given for renewed and more rapid progress of the disease. This, of course, varies exceedingly in different cases.

The *duration* of the disease is generally very considerable, and is always to be counted by years, and sometimes by decades. Even the initial stage may last for twenty years, and there are single cases in literature where patients have lived over thirty and thirty-two years after the first beginning of the disease. With regard to many such cases, it may be said that the duration of the disease cannot be determined; that the malady, having once become stationary, only reaches its end with the natural termination of life, brought about by some intercurrent disease or the weakness of age. This, however, only holds good for a minority of the cases; in the majority of typical cases of tabes, it may be said that the disease puts a limit to the life of the patient in the course of a few years (about six to ten or twelve years), either because its progress within the spinal cord directly threatens life, through bed-sores, cystitis, troubles of respiration, bulbar symptoms, and the like, or because the disease causes a diminished resistance of the individual to intercurrent illnesses (infectious diseases, pneumonia, dysentery, etc.), which bring about death.

Among the *terminations* of tabes, that in *recovery* is certainly the most rare. In quite recent and light cases, in particular, under appropriate treatment, one may sometimes see things change for the better and recovery set in. I have repeatedly seen this take place in the initial stage; to be sure, the doubt may then always arise whether it really was a case of beginning tabes, as long as there was no ataxy yet present. Still, I am in possession of two such cases, which I was obliged with great positiveness to regard as beginning tabes, because the initial symptoms were very distinctly present (lancinating pains, paræsthesiæ, swaying on closing the eyes, weariness and diminished endurance, vesical and sexual weakness, etc.), and which have now been entirely cured for a number of years. But even where the disease is fully developed, we may, although very rarely, see recovery take place, or an improvement bordering on recovery. I am likewise in possession of two cases which demonstrate this. In the one there were lancinating pains, unsteadiness, weariness and distinct ataxy of the legs, paræsthesia in the domain of the ulnar nerve, and vesical weakness. After the malady had lasted for several years, the patient recovered completely, with the exception of slight vesical weakness, and for the past few years has attended to his duties as a civil functionary, without suffering any inconvenience. The other case was that of a naval officer, in whom the disease was farther advanced. He suffered from paræsthesia and slight anæsthesia of the legs, diplopia, distinct ataxy and weariness of the legs; could not walk without being led; had a high degree of vesical weakness (incontinence, with cystitis), etc. After systematic treatment for two years, he was so far restored that he marched for three or four hours, could retain his urine for five or six hours, showed no disturbance of sensibility, got married, and has now for two years served as commander of an iron-clad frigate.

I could make still further reports of similar, if not as well-marked instances of improvement. Since Remak's celebrated case, other observers also have now and then been able to report cures of actual tabes.

There cannot, therefore, be the slightest doubt that in a certain, though very small per cent. of all cases of tabes, recovery actually follows.

Far more frequently patients must content themselves with a slight and *moderate improvement*, or an *arrest of the malady;* and this, too, is a result which does not constitute the rule, but is, likewise, only reached in a certain number of cases.

As a rule, however, this trouble is of a decidedly progressive character, its tendency being *slowly to grow worse.* The final *fatal termination* may then be brought about in various ways, as follows:

The disease in its progress may lead to paraplegia, to cystitis and bed-sores, with their results, and the patient then dies from increasing cachexia under the picture of severe spinal paralysis. In that case we usually see a very protracted death-struggle, and not rarely, during the last days, severe cerebral symptoms, coma, delirium, etc., under which life is extinguished.

Or, in its progress upward, the disease leads to symptoms which directly threaten life, to disturbances of respiration, suffocative attacks, difficulty of swallowing, etc., and thus causes death.

Or, finally, some intercurrent disease (typhus, pneumonia, diphtheria, cholera, pulmonary phthisis, etc.) brings about the end. It is remarkable that so many tabes patients succumb to typhus; still this might in part be explained by the fact that their long sojourn in hospitals more frequently exposes such patients to the danger of infection.

Theory of the Disease.

We have here but little to say on this point. By the theory of tabes we understand not merely the theory of ataxy, which is but a single symptom of the same, but *the reference of the whole symptom-picture to definite anatomical changes in the spinal cord*—the demonstration that a certain definite method of development and form of symptoms stand in a causal relation to a certain definite kind and localization of anatomical changes.

We have been obliged in a number of places to point out the fact that we are, as yet, still far from having reached this goal; we still lack the positive evidence of a whole array of single facts which belong to a theory of the disease. We may therefore here only briefly touch upon those views which are now current, and upon the most satisfactory hypothesis of tabes, at the same time indicating the gaps which they contain and the objections which, as yet, may be urged against them.

The view of some authors, which was advocated with special zeal by Trousseau, that tabes is merely a functional disease—a pure neurosis, and that the anatomical changes are merely secondary results of this neurosis and not necessary causes of the same, is absolutely no longer tenable in view of the entire unanimity of all more recent anatomical discoveries. Tabes is without doubt an organic disease of the spinal cord.

The general opinion at present is undoubtedly to the effect that *the sclerosis of the white posterior columns is the only essential, and in many cases also the exclusive alteration of the spinal cord in tabes*, and that it is abundantly sufficient to account for all, or at least for the most essential symptoms; and that only a part of the symptoms is brought about through a simultaneous localization of the disease in certain cerebral nerves and districts of the brain.

The advocates of this doctrine explain the lancinating pains by the irritation of the sensitive root-fibres and their virtual prolongations within the posterior columns; the paræsthesias and anæsthesias by disease of the ascending sensitive fibres in the posterior columns; the disturbances of co-ordination by affection of the co-ordinatory tracts presumed to lie within the posterior columns; and they only concede the gradual extension of the process to the neighboring portions of the lateral columns to explain the motor paresis and paralysis which arise late. Various other symptoms, the vesical weakness, the sexual weakness, the trophic disturbances, etc., thus remain silently unexplained; and, altogether, the certainty with which the disease of the posterior columns is made responsible for so many different kinds of symptoms is in remarkable contrast to the perfect uncertainty in which physiologists find themselves, in spite of numerous experiments, with regard to the function of these very posterior columns.

There are not a few serious doubts in the way of accepting this exclusive reference of the tabes symptoms to the disease of the posterior columns. In the most extensive degeneration of Goll's columns the specific tabes symptoms are liable to be regularly wanting; very extensive degeneration of the posterior columns has been observed *without* the symptom-picture of tabes; and although the symptom-pictures of the few hitherto known cases of primary sclerosis of the columns of Goll do not seem to agree among themselves, still they also differ from the symptom-picture of tabes. (Pierret found paræsthesia and slight anæsthesia, difficulty of walking, the impulse to run forward, a high degree of weariness, swaying on closing the eyes, no ataxy. Du Castel, on the contrary, paraparesis, trembling, and amblyopia, but no anæsthesia and no ataxy.)

It has, therefore, been found necessary to admit that Goll's columns have nothing immediately to do with the actual tabes symptoms, but that other portions of the transverse section of the posterior columns must be diseased if the symptom-picture of tabes is to arise. Charcot and Pierret have endeavored accurately to establish the fact that in tabes the disease always first attacks the lateral ribbons of the posterior columns, and that the lancinating pains and the paræsthesias are the expression of this initial disease. If the affection extends from here outward and inwards, then there is ataxy (according to Charcot, the co-ordinatory fibres are supposed to lie in the external halves of the posterior columns, between the inner root-bundles); if the affection extends to the gray posterior horns and the posterior roots, a high degree of anæsthesia is produced; its extension to the lateral columns causes manifestations of motor weakness and paralysis, while its extension to Goll's columns is betrayed by no new symptoms.

This view has something very seductive about it, and is also materially supported by a number of beautiful observations. Perhaps it could be opposed by but one remarkable observation recently reported by F. Schultze.[1] Here there was (probably secondary) degeneration of the external ribbons of the posterior columns, which, it is true, manifested itself only by disappearance of the axis-cylinders, not by proliferation of connective tissue simultaneously with a sarcoma of the corpus callosum; during life there were no symptoms of tabes, especially no ataxy.

It will be seen that, for the explanation of important symptoms of tabes, Charcot himself admits a more or less extensive participation on the part of the gray substance and of the lateral columns. Indeed, according to all that we know of the functions of the spinal cord, the theory of an exclusive disease of the posterior columns can hardly be maintained. Thus, for instance, the retardation of the sensation of pain, which is so frequent, and the partial paralysis of sensation, argue with great probability in favor of a disease of the gray substance. C. Lange believes that the excentric pains are also produced through irritation of the sensitive tracts within the gray substance; the vesical and sexual weakness, the articular troubles and other trophic disturbances, likewise point to a lesion of the gray substance. The motor paresis and paralysis can hardly be explained otherwise than through disease of the lateral columns or of the anterior gray horn, and the oft-cited case of Spaeth-Schueppel only admits of the conclusion that the co-ordinatory tracts of the spinal cord also *do not* lie in the posterior columns.

All these facts favor the view *that the typical form of tabes does not depend exclusively upon disease of the posterior columns of the spinal cord, but that other parts in the vicinity of the posterior columns must also be involved in the disease.*

We would announce the following as for the present the most probable theoretical view of the disease of the spinal cord in tabes, a view which is still to be tested by further investigations. The affection begins in the posterior columns, and probably always in the external portions of the same (régions des bandelettes externes), and thus causes the symptoms of the initial stage; it spreads from here longitudinally and transversely; its extension longitudinally is caused by the advance of the disease from below upward; its extension laterally is at first caused by the fact that a secondary degeneration of Goll's columns is gradually added to the primary affection of the *fasciculi cuneati* [Keilstränge], whereby the peculiar and charateristic gross anatomical picture of tabes is caused. The direct transverse extension of the disease to the neighboring gray posterior horns and the contiguous portions of the lateral columns may explain the later, more severe disturbances of sensibility, the disturbance of coördination, the motor paresis and paralysis, the vesical and sexual weakness. This leaves it still to be more accurately determined to what part, in particular, the lesion must extend in order to produce the single disturbances of function named.

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[1] Centralbl. f. d. med. Wissensch. 1876. No. 10.

We believe that a hopeful field for further investigations and observations is here presented; probably much may still be arrived at by careful attention. We must not be satisfied with the conditions found in the posterior columns, and which readily strike the eye, but must above all things cultivate the methods for the examination of the gray substance, which as yet are very crude; at all events, it is not until then that we shall be able to say, with greater confidence, whether the lesion is confined to the posterior columns or not, and how far it regularly extends beyond this.

The determination of the extent of the lesion, as to space, will be the easier task. Then, however, the more difficult question still remains for solution as to the essential character of the pathological process; whether it is an inflammation or not, whether parenchymatous or interstitial inflammation, etc. We need not travel over this ground again. But it is evident that the theory of tabes cannot be complete until these questions also have been answered.

For the present we can only designate this as a peculiar form of disease with a special tendency to localization in definite portions of the nervous system (posterior columns, certain cranial nerves), and to extension chiefly in certain tracts which, anatomically and physiologically, to a certain degree belong together.

Diagnosis.

If, under the name of tabes, we understand only that form of disease defined and explicitly described in the foregoing pages, then its diagnostic recognition, as a rule, offers no serious difficulties. The typical cases, in particular, are very easy to recognize, and are distinguished, in quite a characteristic manner, from the remaining chronic spinal affections. When in such a case of disease, disturbances in sensitive domains (lancinating pains, paræsthesiæ in the extremities, sense of a tight girdle, sense of weariness, etc.) and symptoms on the part of certain cranial nerves have gone before, when diminished endurance and precision of motor efforts appear, when vesical and sexual weakness are noticed, when objective examination shows a distinct disturbance in the co-ordination of movements, while there is but very slight motor paresis or none at all, when the different kinds of sensation seem to be more or less disturbed, when swaying or closure of the eyes appears, when the reflex action of tendons fails, etc., then the typical form of tabes can be easily and positively recognized.

Nor will it be any less easy to diagnosticate with certainty the Friedreich form of tabes, after the description already given above (page 593).

To be sure, such a clear and significant symptom-picture does not exist in all cases, and one will often meet with serious diagnostic difficulties in those obscure, complicated cases which occur not rarely, in which the affection extends a greater or less distance beyond its usual limits, in cases which are complicated with meningitis, with affections of the gray substance, the anterior and lateral columns. We have already stated repeatedly that transition-forms between the different classes of chronic myelitis are not rare; and we here once more declare that it is of less consequence to force an individual case of disease into a definite diagnostic scheme than to conceive as clear a picture as possible of the anatomical changes actually before one, as to their seat and extent, even though these may not exactly fit into the typical mould of tabes, or of any other form of chronic myelitis. If one holds to this, he will find himself in a comparatively safe position, even over against the irregular, complicated forms of tabes which are difficult to classify.

One of the most difficult matters, and at the same time one of the highest practical importance, is the recognition of *tabes at* *its commencement*, in the earliest stages of its development. We will here merely emphasize the fact that, in this stage, attention is especially to be directed to the lancinating pains—often too little regarded by the patient,—to any symptoms that may be present on the part of certain cranial nerves, to paræsthesias of various kinds, *e. g.*, the sense of a tight girdle, and especially paræsthesia in the domain of the ulnar nerve. It should also be borne in mind that slight swaying on closing the eyes—the only disturbance of sensibility recognizable on careful examination,—that a great sense of weariness and a certain unsteadiness in performing the most difficult and complicated movements, as well as the failure of the reflex action of tendons, that slight weakness of the bladder, dribbling of urine after micturition, and disturbances of the sexual organs, are often, at an early period, noteworthy indications of the severity of the disease which is just beginning. The greater the number of these symptoms that exist at once, the more distinctly, in particular, disturbances of motility and sensibility can be objectively demonstrated, the more probable is the diagnosis of tabes. In this way, also, the distinction is easy between this disease and neurasthenia spinalis, which bears some resemblance to the earliest stages of tabes (compare page 379). At the same time, in not a few such cases of beginning tabes the patient will have to be observed for some time before even a probable diagnosis can be made.

Among the other chronic spinal diseases which may come into question in the diagnosis of tabes, we may speak of the following:

Common *chronic myelitis* (transverse, etc.) will, as a rule, be easily distinguished. Here the picture of the disease is essentially characterized by *paralysis* of all the spinal functions, motor and sensitive paralysis, which generally shows a sharp line of demarcation at its upper limit; no ataxy; on the other hand, quite commonly spasms, muscular tension, contractures, greatly heightened reflex action of the skin and tendons; furthermore, actual paralysis of the bladder, often with cystitis; a tendency to the early formation of bed-sores. And even the initial stage of chronic myelitis will generally be easy to distinguish from the beginning of tabes, by the absence of lancinating pains and of the cranial nerve symptoms, and by the early appearance of paretic symptoms.

There may occasionally be some trouble in distinguishing this affection from *multiple sclerosis*, inasmuch as, when the foci are situated in the posterior columns, tabes symptoms may also arise. On careful observation, however, the diagnosis can generally be easily made, as cerebro-spinal sclerosis presents a picture of disease much richer in symptoms and much more complicated than tabes. The following symptoms may be regarded as particularly significant of multiple sclerosis, and should, therefore, engage our attention: great dizziness, headache, psychical disturbances, early nystagmus, scanning speech (which can be distinguished from the ataxic disturbance of speech of the Friedreich patients); the characteristic tremor on voluntary movement, the pareses, paralyses, muscular tensions, contractures, increased reflex action of tendons, etc., in the lower extremities, which are always present, and the apoplectic attacks.

The Friedreich form of tabes, too, which bears a certain resemblance to multiple sclerosis, on account of the nystagmus, the disturbance of speech, and the static ataxy of the muscles of the trunk, can generally be distinguished from the latter disease on the ground of the symptoms named.

The differential diagnosis between tabes and *spasmodic spinal paralysis* (*lateral sclerosis*), the aggregation of spinal symptoms described by me (compare the following section, No. 13), is very easy; the latter is characterized by paresis and paralysis, wit muscular tension and contractures, with greatly increased refle action of the tendons, and by the absence of ataxy, of disturb ances of sensibility and of the bladder, of lancinating pains, cranial nerve lesions, etc.

The question of distinguishing tabes from *affections of th cerebellum* has greatly occupied authors, and sometimes offers n little difficulty. The evidences of cerebellar trouble are: frequen and severe headache, especially in the back of the head, occa sional vomiting, and general convulsions, a staggering gait, lik that of a drunken man, reminding one of ataxy, but not identi cal with it, sometimes a straddling gait, or an impulse to walk backward, diplopia, and amaurosis. On the other hand, in cere bellar affections there are, as a rule, no lancinating pains, no disturbances of sensibility, no vesical or sexual weakness. By these means a diagnosis can be made in most cases.

As a rule, tabes can be easily distinguished from *chronic spinal meningitis;* in some few cases, however, this cannot be done, because the latter is occasionally complicated with tabes. The evidences in favor of meningitis are the pains in the back which may exist, and the stiffness of the back, the sensitiveness of the spinous processes to pressure, the more or less uniform but not very extreme sensitive and motor paresis, the absence of ataxy, etc.

The difference between *progressive cerebral paralysis* and tabes will be apparent by the absence, in the latter, of the disturbances of speech and psychical changes which are characteristic of the former. Those cases in which the two forms of disease are combined, whether the psychosis is added to the latter history of tabes, or whether symptoms of tabes arise during the course of progressive paralysis, will call for a special exercise of judgment, and will, as a rule, not be hard to interpret aright.

Prognosis.

Tabes dorsalis is, under all circumstances, a very serious disease, although, indeed, its prognosis is not quite so hopeless as it was once thought to be by Romberg. It is certainly not quite as unfavorable as the prognosis of multiple sclerosis, or even as that of simple transverse chronic myelitis.

Although in the great majority of cases the disease is more or less continuously progressive, advancing to an unfavorable termination, still a certain number, even though it be but a small number, of cases end in recovery. In many instances at least a certain degree of improvement takes place, and the malady remains at a stand-still for years. If the disease is at all well developed, it is hardly to be supposed that the anatomical changes can recede.

It is always a question of a disease of long duration—and this is at least a comfort to many patients; however variable it may be in individual cases, a number of years always elapse, often many, very many years before the fatal termination. The slower the development of the disease, the better the general condition, the slighter the disturbance of sensibility, the less there is of a neuropathic predisposition, the longer, as a rule, will be the duration of the disease.

The judgment of each individual case, with regard to the prognosis, is generally very difficult; even one who has a rich experience in such matters will be able to give but an uncertain verdict. The prognosis is *favorably influenced* by the absence of a neuropathic predisposition and hereditary causes, by the absence of previous sexual excesses, by a very slow development and moderate intensity of the symptoms, by but slight disturbances of sensibility, rare and moderate lancinating pains, by the favorable effect of certain therapeutic measures, by favorable external circumstances which enable the patient to do everything requisite for his health, etc. On the other hand, it is *unfavorably influenced* by hereditary predisposition, sexual overstimulus, by the rapid progress of the manifestations, severe paroxysms of pain, rapid emaciation and loss of strength, by affections of the special senses and of the brain, great general irritability, serious irritation of the genitals, a tendency to bedsores, vesical catarrh, by the uselessness of all therapeutic interference, by unfavorable circumstances in life, forcing one to labor for his daily bread, etc.

The *prognosis of individual symptoms* is very various. While the disturbances of sensibility, the paræsthesias and anæsthesias, are generally very easily improved or removed, the prognosis of the ataxy is ordinarily quite unfavorable; it generally remains unimproved, even though the motor power as a whole improves. The lancinating pains, as a rule, are extremely obstinate; the vesical weakness may disappear, the sexual weakness usually remains unimproved. Paralysis of the muscles of the eye generally admits of a favorable prognosis, while the amaurosis due to atrophy of the optic nerve is almost absolutely hopeless; it is almost always incurable, and but rarely can even any slight improvement be observed.

Therapeutics.

With regard to the *prophylaxis* of tabes, which ought perhaps to be considered oftener than it is now, we may refer to what was said above on the therapeutics of chronic myelitis. The points to be specially noted in this connection are the combating of the neuropathic tendency by appropriate diet and habits of life, and the prevention, by every means in our power, of things notoriously injurious, especially the combating of onanism and avoidance of sexual excesses. In not a few families will one find fitting objects for the exercise of this sort of household medical supervision.

In most cases it will be found impossible to fulfil a *causal indication*, as one is dealing with quite chronic conditions, the causes of which have long since passed away. We shall, therefore, but rarely find ourselves in a position to do anything in this direction, and whatever is to be done will appear from the individual circumstances of the case. Where there are signs of syphilis, we should not fail to carry out the appropriate treatment.

As a rule, the physician will find that he has to deal with the disease after it has arrived at a certain degree of development, and he will have, before all else, to satisfy the *indicatio morbi*. What we have said more specifically above, under the therapeutics of chronic myelitis, is here especially applicable. At the same time, we must not fail just here especially to emphasize some facts, and again to subject to closer inspection the effects of certain remedies in tabes particularly. Of all forms of chronic spinal affection, tabes is certainly the one that has been subjected to the most extensive and manifold therapeutic experiments.

As a rule, nothing at all is to be expected from actual *antiphlogistic* treatment. This could at most be thought of in quite recent cases, with well-marked evidences of irritation and a more inflammatory character; and here, according to the observations of Frerichs, it does sometimes seem to be useful. But in all later stages, and during the well-marked chronic course of the disease, antiphlogistic treatment has always proved useless. As a rule, therefore, it is to be ignored. But the application of dry cups may often be permitted for the relief of pain.

In just this form of chronic myelitis *derivatives* also seem to accomplish the least possible good. This is especially true of the actual cautery, which was formerly very often used, but is to be discarded as entirely useless. I have likewise never seen any distinct benefit from the milder derivations (blisters, salves that raise pustules, irritating inunctions, etc.).

Among the means of treatment still discussed for tabes, *simple thermal baths* formerly headed the list. If anything is plain in the therapeutics of tabes, it is the fact, confirmed almost unanimously by all recent experience, that *thermal baths are injurious rather than beneficial in tabes*, that in this form of chronic myelitis they should either be entirely avoided or only employed with the utmost care. Benedict says, outright: "Thermal baths belong to the essentially injurious agencies in tabes;" and M. Rosenthal, Erdmann, von Krafft-Ebing, Richter, and others, have expressed themselves quite to the same effect; and my own experience likewise quite agrees therewith. Leyden is the only one among recent writers who expresses himself as "unequivocally" in favor of the value of thermal baths. In opposition to this view, we would maintain what was said generally above (page 460) on the subject of thermal baths, under the head of Chronic Myelitis, as being especially applicable to tabes. We would advise the trial of thermal baths in tabes patients only under quite peculiar conditions, when all other methods of treatment have failed. The cases which are generally declared to be especially adapted to the use of thermal baths are those with prominent evidences of irritation, lancinating pains, with great general excitability, sleeplessness, etc. It is very much to be wished that these indications might be made more exact.

At all events, one should be extremely cautious in the application of thermal baths. Only a moderately warm temperature is allowed; all above 90° F. may be injurious; most of the natural thermal waters therefore must be cooled. The duration of the baths must not be too long (not over fifteen to twenty minutes); they are not to be employed daily, but only every two or three days. Only when thus carefully used do thermal baths sometimes seem to be of use in tabes.

The *sulphur baths* which have been used with special frequency in France have probably no other effect than that of simple thermal baths; what has just been said, therefore, applies equally to them.

Saline thermal baths can certainly show better results, and Rehme, in particular, has long enjoyed a well-founded reputation in the treatment of tabes. From my own experience I can likewise make a report in the main favorable to these baths (Nauheim). But here, too, it is greatly to be desired that more accurate indications and contra-indications should be established. The rules of caution given above (p. 461) might be especially applicable to tabes; the baths should not be given too often (Waldmann considers three baths a week as sufficient for most cases), they should not be of too long duration nor too warm, and the more exciting forms of baths should be especially avoided, as they only too often interfere with the good results to be expected from such a course of baths.

No conclusive experience is at hand with regard to the effects of *chalybeate-baths* or *mud-baths* in tabes. The latter seem to act favorably in some cases, though we cannot as yet say precisely in what class of cases.

But a *cold-water cure*, carefully conducted, must be very especially recommended for the treatment of tabes. The results thereof are extraordinarily favorable, especially in comparison with the other curative measures at our command. Aside from the hydropaths, whose judgment might be impugned as not altogether unbiassed, Benedict, M. Rosenthal, and others, express themselves decidedly in favor of the beneficial action of hydrotherapeutics. Waldmann ascribes a strikingly favorable effect to mild, cool sponging of the body. Here also Leyden alone sets himself in opposition to all other authors, and claims that tabes patients, as a rule, bear cold water badly. According to my own experience, I must directly contradict this statement. Among nineteen of my tabes patients who went through with the cold-water cure, sixteen experienced more or less benefit, two saw no improvement, and only one grew slightly worse. Most of my patients bear cold-water treatment so well that I almost always have them use the cold sponge-bath at home all winter, and have never yet seen any harm come from it.

Here of course everything depends on the method and the manner of application, which must be determined and regulated with the greatest caution.

The method of action of the water-cure on tabes may be two-fold. In the first place, it contributes to the hardening of the skin and thereby to the diminution of injurious effects; it causes general strengthening and improvement of nutrition, which then, secondarily, also effects an improvement of the spinal trouble; One almost always sees patients leave water-cure institutions with a blooming appearance, increased weight, and a feeling of greater well-being and increased capacity for work; and all this is certainly not to be lightly esteemed.

But, secondly, the water-cure seems also to exert a direct and beneficial influence on the affection of the spinal cord, and thus to contribute more directly to the improvement and cure of the disease.

From the statements of various specialists with regard to the *methods* to be selected, it first of all appears very decidedly that all low temperatures as well as all more exciting forms of bath are injurious and to be avoided, that, on the contrary, moderate degrees of temperature—not below 68° F. !—and the milder, more quieting, or only slightly exciting forms of bath alone are permissible. While, therefore, actual *cold* water, cold full-baths, shower-baths, cold slappings, etc., are decidedly forbidden, it is admissible to use simple wet rubbings (beginning with 77° F. and going down to 68°, or at the utmost to 59°), half-baths (of about 86° to 70° F.) with simultaneous washing of the back and sprinkling of the back; furthermore, also mere washings of the back, of the feet, wrapping the feet and the abdomen with cold compresses which warm themselves, and here and there a hip-bath. The wet pack should be used with extreme caution, beginning with a temperature of 88° F.; it is seldom borne at a temperature below 77° F. (F. Richter). Czerwinsky recommends only tub-baths to be used in tabes, with a temperature of 81½° F., which is to be lowered only very gradually. At the same time, some patients bear lower temperatures very well. Very much reduced, anæmic, irritable patients, who are very sensitive to cold, are the least fitted for this treatment; at all events, in such cases it may only be tried with the utmost caution.

The choice of the institution to which tabes patients are to be entrusted is of the greatest importance. It is not in all water-cures that the requisite understanding and the necessary individualization are to be found. Care must therefore be exercised in this respect.

If possible, let those institutions be selected which lie in a good mountain region, are provided with convenient walks, are well conducted as to attendance, and are not too noisy. The best plan is to send patients there at the beginning of summer with instructions to remain till autumn. I have thus far not found winter-cures in actual cold-water institutions as especially to be recommended.

Since the pioneer labors of Remak, the *galvanic current* has earned for itself a secure and honored place in the treatment of tabes, as well as in that of other diseases. Electro-therapeutic literature abounds in reports of cases which establish, beyond all question, the value of the galvanic current in tabes. My own experience also speaks sufficiently in favor of this good effect, although it is certainly calculated to put me on my guard against too great illusions in regard to the value of the method. Among 66 cases treated by me, 25 received no benefit; 41, on the contrary, were more or less improved. This improvement, in most cases, it is true, did not amount to very much; in but comparatively few was it very considerable, and only in rare instances was there complete recovery.

Various authors differ somewhat among themselves with regard to the methods which are to be employed, but almost all agree in this, that they regard the main point as consisting in the direct treatment of the back, with moderately strong, chiefly stabile, more rarely labile currents, and recommend only short sessions.

Von Krafft-Ebing advises simple stabile currents through the spinal column, of from four to six minutes' duration; in addition, he recommends the labile action of the cathode on the nerve-trunks. I cannot coincide with his opinion, that usually the first six or eight sessions determine the result; the improvement often does not appear until very much later. Mendel has likewise applied stabile and labile currents to the spinal cord with advantage. Mor. Meyer has found the action of the anode useful in some cases, on points in the spinal column that were painful on pressure.

I myself for some years past have almost exclusively employed the method described on p. 183, and believe that I have thereby attained comparatively the best results. Naturally this will have to be variously modified in different individuals, so far as the duration, intensity, and localization of the currents are concerned. In very irritable persons, in particular, with severe pains, it is well to apply very weak currents. I generally combine with the galvanic treatment of the spine peripheral galvanization of the nerves of the lower extremities (cathode labile), which is to be used about two or three times a week. In the same manner, any complications that may exist (paralysis of the muscles of the eyes, atrophy of the optic nerve, lancinating pains, vesical weakness, anæsthesia, etc.) are at the same time to be treated symptomatically.

The sessions last about three to six minutes ; strong currents are positively avoided ; treatment usually takes place daily, commonly for months together. We should not allow ourselves to be prevented from continuing the treatment by temporary fluctuations in the course of the disease.

Only if patients feel more tired and poorly after each galvanization ; if their condition, as a whole, grows gradually worse ; if their pains increase, sleeplessness sets in, or the like,—must we give up the galvanic treatment as inappropriate.

Among the *internal remedies* for tabes, *nitrate of silver* undoubtedly stands first, as it can show quite undoubted results. The observations of Wunderlich, Charcot, Vulpian, Eulenburg, Griesinger, von Graefe, and others, speak decidedly in its favor, and lately Friedreich has also again expressed himself favorable thereto, at the same time pointing out the danger of albuminuria connected with its use ; but this danger may no doubt be easily avoided by cautious dosing.

Although we cannot, as yet, accurately designate the cases in which nitrate of silver proves beneficial, still the fact that it really [illegible] done good in a considerable number of cases, and that, [illegible] f used with some caution, its employment is without any danger or discomfort, is of itself enough to justify its administration in most cases. The dose is 0.01 to 0.02 gramme (one-sixth to one-third of a grain) several times a day, so that *at most* 0.06 to 0.10 gramme (one to one-and-a-half grains) is used in the course of a day; the remedy may be continued until about 8 to 10 grammes (two to two-and-a-half drachms) have been used; and its use may also be repeated again later.

Caster found material improvement under the oxide of silver in two cases where the nitrate had been employed without effect.

Iodide of potassium, which is recommended by some on theoretical grounds, cannot show any results worth mentioning. Leyden considers it effective against any meningitic complications that may exist; it is also said to moderate symptoms of irritation.

According to Siredey, *bromide of potassium* is said to improve the disturbances of co-ordination and to mitigate the pains in doses of from 3 to 10 grammes (gr. xlv. to ʒ iiss.) daily.

The systematic use of *belladonna* and *ergot* has, as a rule, only resulted in failure. Ergot has lately found a eulogist in Waldmann, who tried it on himself (he suffers, however, principally from spinal meningitis), and who urgently recommends it for chronic meningitis and for tabes "caused by dilatation of the vessels." One to two grammes (fifteen to thirty grains) is to be given daily, in the form of. powder, and continued for a considerable time.

There is not much to be said in praise of *arsenic*, *chloride of gold and sodium* or *chloride of barium* (recommended by Hammond in doses of 0.05 gramme [three-fourths of a grain] three times a day); all these remedies generally leave one in the lurch.

Strychnine is decidedly objectionable in tabes.

Phosphorus, which has repeatedly been recommended by Dujardin-Beaumetz, appears to produce no effect in most cases, and will but rarely be used on account of the danger connected with it.

Quite recently, in a Russian periodical, Lesch has reported favorable results from phosphorus treatment in tabes, from the clinic of Prof. Eck. The remedy is said to act first on the sensibility, afterwards on motility also. Five centigrammes (three-fourths of a grain) of phosphorus is to be dissolved in sulphuric ether, and this is to be made into fifty pills, with breadcrumbs; three to six pills are to be given daily, until about 0.15 to 0.25 gramme (two and a half to four grains) of phosphorus have been consumed. I tried the remedy in one case, without any result; it was well borne. Further trials are to be desired.

Cod-liver oil is praised by many. It appears, besides its general nourishing qualities, also to have a favorable effect on the nutrition of the nervous system, and therefore deserves to be used where it is well borne.

Very particular attention is, in all cases, to be given to the matter of *regulating the diet and the method of life* of tabes patients. On this subject we may refer to what was said under Chronic Myelitis (see above, p. 464) and also to the admirable remarks of Waldmann on this point. We have nothing to add to what we have said above with regard to diet, mental labor, digestion, sleep, sexual intercourse, etc. We would again recommend the utmost possible indulgence in fresh air, especially mountain-air, sea-air, and the air of the forests. As a feeble substitute for a winter spent in the south, which is impracticable for many patients, the daily inhalations of oxygen, lauded by Waldmann, might be tried.

With reference to bodily movements, we may, according to our best convictions, dissuade patients from too much walking. The diseased spinal cord must be spared; patients quickly grow tired in walking, and may, therefore, only continue this exercise until the beginning of fatigue; every overexertion or extreme fatigue may be of the greatest detriment. Topinard likewise recommends rest, especially for those people who otherwise work hard and beyond their strength; on the other hand, he thinks that light muscular exertion, though never to the point of overexertion, is the rather indicated for others, especially patients from the higher walks of life, who have a poor appetite and lowered nutrition. The proposition to cure commencing tabes by quiet rest in bed for several months has not met with any endorsement. As a matter of course, tabes patients must guard themselves against wet, dampness, cold, and taking cold; for many of them it is therefore well to wear flannel underclothing.

After having thus passed in review the different means and methods recommended against tabes, it may be well to offer a few suggestions with regard to the *general plan of treatment* which should be carried out in individual cases.

In *cases just commencing*, with light, initial symptoms, we should first search for any causes of the disease which may be present and still acting, and remove these. Then the diet and method of life of the patient should be regulated in the most careful manner, his activity in his business, his recreations, etc., and the patient should early have his attention called to the fact that only the most methodical and persistent carrying-out of the regulations prescribed for him may preserve him from a subsequent grievous malady. Of course, we should at the same time strive to quicken his hope and courage. Much must still be left to the tact of the physician, after careful weighing of the individual characteristics of the case. Direct treatment in summer may be limited to having the patient take up his abode for a few weeks at some appropriate place in the mountains, in good forest air, or at the sea-side, to having him use a regular cold-water cure, or a cure at the baths of Rehme or Nauheim. We may generally reserve for the autumn and winter the regular carrying-out of systematic galvanic treatment, the administration of nitrate of silver, or any other medicine called for by circumstances, as well as the regular use of cold rubbings, washing of the feet, of the back, etc. In this way different therapeutic measures may be carried on for months and years, interrupted from time to time by pauses. If improvement or recovery takes place, patients must still take good care of themselves for a very long time, avoid all overexertion, and endeavor permanently to establish their health and strength by subsequent treatment (mountain-air, water-cures, sea-baths, etc.).

In quite recent cases, with very prominent signs of irritation, Frerichs (compare the dissertations of Fabricius, Kauert, Mette, Nachtweyh) recommends the copious abstraction of blood along the spinal column, and derivation to the skin and bowels by flying blisters, irritating salves, and colocynth. This method may only be employed with the greatest caution, and is, of course, entirely to be rejected in the later stages of the disease.

In more advanced and *completely developed cases* we shall not be able to build any too great hopes on therapeutics, however much it may be our duty to support the hopes of the patient. There will be plenty of chance here for the systematic trial of various means of cure. For the summer months a course of baths at Nauheim or Rehme may be recommended; afterwards also at some one of the thermal springs, with subsequent residence in the mountains, or a cold-water cure. In the intervals the patient should be allowed a couple of weeks of rest at a pleasant place that suits him, and without any special treatment, except perhaps cool rubbings. For the winter and spring months the use of the galvanic current especially commends itself, simultaneously with internal means and cautious hydropathic treatment. If the use of nitrate of silver has been continued long enough, we may try, one after another, the internal remedies mentioned above, giving tonics, cod-liver oil, and the like, in the intervals. The method of life must be regulated; care must be taken that patients remain in the open air as much as possible; they should use a good perambulating chair, and we must seek by all sorts of external means, by rubbing with liniments and spirituous washes, by symptomatic treatment, to act favorably upon the state of mind of the patient. All this is a very difficult task for the physician, and patients only too readily lose courage and turn from one physician to another, expecting help from each one, and dismissed by each with the same lack of result.

In *quite old, incurable cases*, finally, we should seek, so far as possible, to restrain patients from all useless attempts at curative treatment. Romberg's utterances, which have become celebrated, and were dictated by true humanity, apply to these cases. If patients possess enough strength of character, they should be cautiously informed of the uselessness of further "cures;" if not, we may endeavor to keep them encouraged, which is generally very easy, with the hopes of improving at a better time of year, and conduct them through their long period of suffering with harmless treatment. We should then confine ourselves to regulating the method of life and the external relations as far as possible, seeking to secure to the patient as comfortable an existence as may be, giving him careful symptomatic treatment, and letting him spend much time in the open air, at the same time improving the general strength by cold rubbing and sponging. Long journeys to bathing-places are decidedly to be avoided; still we may advise well-to-do patients, who can accomplish it with all comfort to themselves, to spend the summer where they can have good mountain or forest air.

In so tedious and severe a malady, running its course in part with very tormenting symptoms, the *symptomatic indications* often urge themselves upon the physician. One must be very careful and ingenious in order to satisfy the desires of the patients.

First on the list are the *pains*, especially the *lancinating pains*, the paroxysms of pain, by which patients are often terribly tormented. Only too often we shall find ourselves helpless over against them, endeavoring in vain to combat them with every possible means. I here mention a list of remedies, all of which help sometimes, but quite as often leave us in the lurch: sinapisms, blisters, warmth (in the form of hot compresses, sand-bags, etc.), Priessnitz's cold-water compresses, opium or belladonna plasters, rubbing with chloroform, veratrine, or oil of hyoscyamus; faradization or galvanization (stabile cathode) of the particularly painful and hyperæsthetic points of the skin; but, above all, subcutaneous injections of morphine, which only too readily become a necessity; among other internal remedies, quinine and bromide of potassium in large doses, or the hydrobromide of quinia, recently recommended by Erlenmeyer (0.50 to 2.0 at a dose, gr. viiss. to gr. xxx.); iodide of potassium (Leyden) in moderate doses, etc.

Against *cutaneous anæsthesia*, *motor weakness*, and *atrophy of the muscles*, electricity is the only rational means to be employed.

In *weakness of the bladder*, faradization of the bladder, either from without or with the aid of the bladder electrode, is often useful.

Cystitis and threatening or existing bed-sores are to be treated in the manner explicitly laid down before (p. 194).

For the *amaurosis*, strychnine has always thus far been tried in vain; the galvanic current, also, is extremely seldom of use. We may deem ourselves fortunate if we accomplish the arrest of the evil while still in progress of development.

The *muscular twitchings* that are sometimes present demand the same means as were indicated for the pains.

The *constipation*, which almost always exists, is an especially troublesome symptom. We must proceed against this carefully, and only with the mildest purgatives. If we can succeed in attaining our end by appropriate diet (much fruit, Graham bread, etc.) and simple enemas, so much the better. In specially obstinate cases the regular faradization of the intestines (one pole to the sacrum or in the rectum, the other passed over the entire abdomen, with a strong current) often yields good results.

Attacks of *oppression* must be combated by the customary means. *Paroxysms of gastralgia* are best met with a strictly limited diet and morphine injections.

The best remedy for the *pollutions* and *increased sexual excitability* is bromide of potassium (gr. xxx. to gr. xc.). Lupulin and camphor have also often proved useful. Cool hip-baths, the avoidance of any considerable filling of the bladder, and of lying on one's back at night, are also of benefit.

13. *Paralysis Spinalis Spastica.—Tabes Dorsal Spasmodique (Charcot).—Sclerosis of the Lateral Columns?—Primary Lateral Sclerosis?*

Tuerck, Ueber primäre Degenerat. einzelner Rückenmarksstränge. Sitzungsber. d. k. Akad. d. Wissensch. zu Wien. Math. naturw. Classe. Bd. XXI. Jahrg. 1856. S. 112.—*Charcot*, Sclérose des cordons latér. d. l. moelle ép. chez. une femme hystér. atteinte de contracture perman. des quatre membres. Gaz. hebdom. 1865. Nr. 7.—*Charcot* et *Joffroy*, Deux cas d'atrophie muscul. progress. avec lésions de la subst. grise et des faisc. antéro-latéraux de la moelle ép. Arch. d. Physiol. II. pp. 354, 629, 744. 1869.—*A. Voisin*, Meningo-myëlite suraigue, Sclérose des cordons latéraux, etc. Gaz. méd. de Par. 1869. Nr. 40. p. 538.—*Gombault*, Sclérose symmetr. des cord. latér., etc. Arch. d. Physiol. IV. p. 509. 1871–72.—*Charcot*, Sclérose primit. de la part. postérieure des cordons antéro-lat. Gaz. méd. de Par. 1874. Nr. 3. pp. 38–39.—*Charcot*, Leçons sur les mal. du syst. nerveux. II. Sér. fasc. 3. Sclerose latérale amyotrophique. Paris, 1874.—*W. Erb*, Ueb. einen wenig bekannten spinalen Symptomencomplex. Vorl. Mitth. Berl. klin. Woch. 1875. Nr. 26; und Bericht üb. d. 8. Versamml. südwestdeutsch. Irrenärzte zu Heppenheim. Zeitschr. f. Psych. Bd. 32. 1875.—*Fr. Richter*, Zur Sklerose der Seitenstr. des R.-M. Deutsch. Arch. f. klin. Medic. XVII. S. 365. 1876.—*O. Berger*, Die primäre Sklerose der Seitenstränge des R.-M. Deutsche Zeitschr. f. prakt. Medic. 1876. Nr. 16–19.—*Seeligmueller*, Sklerose d. Seitenstr. d. R.-M. bei versch. Kindern derselben Familie. Deutsche med. Woch. 1876. Nr. 16-17.—*Leyden*, Klinik der Rückenmarkskr. Bd. II. S. 434. 1876.—*O. Berger*, Ein Fall von Sclerosis later. amyotroph. Deutsche Zeitschr. f. prakt. Med. 1876. Nr. 29, 30.—*Bétous*, Étude sur le tabes dorsal spasmodique. Paris, 1876.—*Charcot*, Du tabes dorsal spasmodique. Progrès médic. 1876. Nov. Nr. 45-47.—*Erb*, Ueber Lateralsklerose und ihre Beziehungen zur Tabes dorsalis. Arch. f. Psych. und Nervenkr. Bd. VII. S. 238. 1876.—*Berger*, Zur Lehre von der primären Lateralsklerose des R.-M. Deutsche Zeitschr. f. prakt. Medic. 1877. Nr. 3, 5, u. 6.—*Rich*, *Schulz*, Mehrere Fälle von "Lateralsklerose." Arch. d. Heilk. XVIII. S. 352. 1877.—*W. Erb*, Ueber spastische Spinalparalyse (Tabes dorsal spasmodique, *Charcot*). Virch. Arch. Bd. 70. 1877.

Introduction and History.

It should be stated, in advance, that in this section we shall consider that form of disease exclusively, the picture of which I first gave fully and in detail (Berliner klin. Woch., 1875, No. 26), and which Charcot has also recently described in detail under the name of "tabes dorsal spasmodique." This was after that author had for a number of years made short and suggestive reports with regard to quite similar manifestations of disease, and had spoken of them as being related to sclerosis of the spinal lateral columns.

In the meantime, the opinion, that sclerosis of the lateral columns is the anatomical basis of the disease described by Charcot and myself, has been gaining more and more advocates; indeed, it is held by some authors as quite firmly established, although this is without sufficient grounds, that is, without conclusive anatomical investigation.

It is only on the supposition that this opinion, which we ourselves regard as extremely probable, is correct, and will be confirmed by subsequent investigations, that we also discuss sclerosis of the lateral columns in this section, that in the historical review we allude to those works which treat of *primary* degeneration of the same, and that we shall depict its anatomical characteristics. If, contrary to our expectation, this opinion should prove to be false, the disease here to be described would nevertheless remain as an independent, well-characterized form of disease, and we should still have to search for the symptom-picture of sclerosis of the lateral columns.

Tuerck was doubtless the first who recognized a primary sclerosis of the lateral columns; he expresses himself only briefly and unsatisfactorily with regard to its symptomatology.

All other communications up to 1875 actually originate with Charcot alone. The manifold investigations into secondary degeneration of the lateral columns, which fall within this period, at most furnished a few fixed points in symptomatology; and these are capable of but very limited application, on account of the primary disease of the brain or spinal cord.

Charcot first published a case of hysterical contracture which had lasted continuously for nine years, and had before arisen repeatedly; the autopsy showed symmetrical sclerosis of the posterior sections of the lateral columns throughout the greatest portion of their length. Subsequently, Charcot set up a peculiar form of lateral sclerosis, which is complicated with muscular atrophy, and which he afterwards designated as "sclérose latérale amyotrophique;" in giving the description of two cases belonging under this head, he briefly defined the essential symptoms of sclerosis of the lateral columns. His subsequent communications always brought merely brief references of essentially the same import.

The matter then stood thus: The principal symptoms of sclerosis of the lateral columns had little by little been recognized, being taken from various complicated cases (secondary degenerations, amyotrophic lateral sclerosis, etc.); and from these the picture of "primary lateral sclerosis" had been constructed, with great probability of correctness—this being also said to have been found present in some of the cases observed by Charcot. These cases, however, were never published; no detailed disease-picture of lateral sclerosis was anywhere delineated; the disease figured in no text-book; and, at least with us in Germany, it did not occur to anybody to recognize and diagnosticate this as a very frequent and well-characterized form of disease.

My introductory communication gave the delineation—containing all that was essential—of a disease-picture which bore the greatest resemblance to the symptom-picture of lateral sclerosis indicated in outline by Charcot, which may probably be regarded as identical with the latter, and actually is so regarded by most authors.

Charcot himself, then, in 1876, both had this disease described by his pupil Bétous, under the name of "tabes dorsal spasmodique," and also himself published an essay thereon. He likewise, as yet, expresses himself very guardedly concerning the anatomical basis of the disease, which can only be finally determined by renewed anatomical investigations. He says that the cases formerly observed by him are too old and partake too decidedly of the character of "somewhat confused memories" to serve as evidence.

During the past year several works have also appeared in Germany on so-called lateral sclerosis, and O. Berger, in particular, has handled the subject in several essays. I have myself recently published my more detailed communications on this disease.

Definition.

The disease is *clinically* characterized by a gradually increasing *paresis* and *paralysis*, generally advancing slowly from below upwards, with *muscular tension*, *reflex contractions* and *contractures*, with strikingly *increased reflex actions of tendons*, while at the same time there is entire or almost entire *absence of all disturbances of sensibility or trophic disturbances*, of all *vesical or sexual weakness*, and of all *cerebral disturbances*.

The disease, as a rule, is extraordinarily slow in its development, insidious and very chronic in its course; it may occur in various combinations, but in its typical cases is very easily to be distinguished from all other forms of chronic spinal disease (from chronic myelitis, myelitis transversa, tabes dorsalis, multiple sclerosis, etc.).

The *anatomical* characteristics of the disease cannot as yet be given with certainty; if everything does not deceive us, the anatomical basis of the group of symptoms consists in a symmetrical sclerosis of the lateral columns, especially of their posterior divisions, advancing gradually from below upwards. It would constitute a complete analogue to sclerosis of the posterior columns. The correctness of this presumption can only be definitely decided by post-mortem examination.

The *designation of the disease* would naturally be best selected on an anatomical basis, and "sclerosis of the lateral columns" or "lateral sclerosis" would therefore be most appropriate. As long, however, as we are not certain in this matter, it would be better to select a symptomatic designation. Charcot has chosen the term "tabes dorsalis spasmodica." I cannot consider this choice very fortunate, and it is hardly to be accepted for us in Germany. For us the term "tabes dorsalis" now signifies exclusively sclerosis of the posterior columns, and it is doubtless best for us to abide by it. But if by the term tabes we are again to designate all atrophic and sclerotic processes in the spinal cord, we should thereby still be in advance of our anatomical knowledge of the disease. Furthermore, in Charcot's designation the spasmodic element of the disease is the only one that receives due prominence.

But the main symptoms of the disease are manifestly *paralysis* (paresis) and *spasm* (muscular tension and contractures); these should, therefore, receive special prominence in the designation. After various other trials—following the analogy of atrophic spinal paralysis—I have chosen the name "spasmodic spinal paralysis," "paralysis spinalis spastica," as a provisional clinical designation, and hope that, in spite of its lack of euphony, it will suffice as a somewhat suggestive name for the disease, until satisfactory results of post-mortem examinations shall render possible the choice of a definitive anatomical designation.

Etiology and Pathogenesis.

There is thus far very little to be said on this point. The disease is a tolerably frequent one, although by no means so frequent as sclerosis of the posterior columns. But little is known of any definite *predisposition* thereto. Only in the smallest number of cases can the *neuropathic tendency* be assumed as an auxiliary cause. The disease seems to attack the *male sex* somewhat oftener than the female; still the difference is decidedly not so great as in tabes.

The disease is developed almost exclusively during the *age of maturity;* by far the larger number of cases begin between the ages of thirty and fifty. But from various observations which I have reported (l. c.), it would appear that the disease may also develop itself even *in earliest childhood,* and we might then imagine the possible congenital defective development of certain divisions of the spinal cord. Part of the cases described under the name of spasmodic infantile paralysis may well belong here. The remarkable observation communicated by Seeligmueller, where four children of the same family were taken sick with amyotrophic lateral sclerosis, should also be placed here.

But most frequently it is just the robust, strong people, who are subject to no neuropathic influences, that are attacked by the disease, and in the majority of them the most careful interrogation does not discover any sort of predisposing cause for the disease.

In fact, even the *exciting causes* of spasmodic spinal paralysis are as good as unknown. Naturally enough, here, too, *taking cold* is not rarely assigned as a cause, although not so often, nor with as much certainty, as it is in some other things. At the same time, the astonishing many-sidedness of this source of injury may occasionally show itself in this malady too.

Bétous thought that he could demonstrate *lead-poisoning* as a cause in one of his cases. Berger believes in the possibility of its being produced after *traumatic influences.* In my cases I could never prove anything with regard to the influence of *sexual excesses* or previous *syphilis.*

The etiological relations of the disease, therefore, certainly still remain to be discovered.

Pathological Anatomy.

Going on the supposition announced above, and which is to be still further established below, that the disease is identical with primary sclerosis of the lateral columns, we have here briefly to depict the anatomical conditions in this affection.

Macroscopically we encounter the usual appearance of gray degeneration, though here limited in a symmetrical manner to the two lateral columns, and, indeed, especially to the posterior divisions of the same. The gray discoloration here occupies the entire posterior half of the lateral column in the form of a triangle, extending inwards to the posterior gray horn, outwards to the pia mater, and being somewhat indistinctly shaded off forwards into the healthy tissue of the lateral column (Fig. 11, *a*). According to Charcot and Bouchard, this very form of the gray wedge alone should enable one to distinguish primary lateral sclerosis from secondary degeneration in most of the sections of the cord. Secondary degeneration of the lateral columns, as a result of disease of the spinal cord, is characterized by the fact

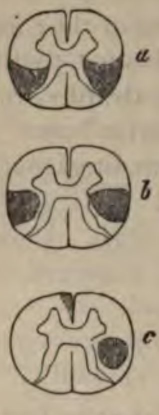

FIG. 11.—Half diagrammatic representation of the various forms of sclerosis of the lateral columns (from about the upper part of the dorsal region). *a.* Form of gray discoloration in *primary* sclerosis of the lateral columns. *b.* In *secondary* degeneration as the result of disease of the spinal cord. *c.* In secondary degeneration as the result of disease of the brain.

that the inner posterior contour of the gray spot does not reach the posterior horns, but remains separated from it by a narrow zone of normal white substance, while the gray discoloration, it is true, here also extends to the pia mater (Fig. 11, *b*). Secondary degeneration, the result of brain trouble, in addition to its usually appearing only on one side, is said to be distinguished by the fact that the gray discoloration has a more roundish form, standing out from the surrounding substance in sharp outlines, and never reaches close to the pia mater (Fig. 11, *c*). Still these characteristics are probably not absolutely universal, though they may, perhaps, apply to most cases, and to the greater part of the spinal cord.

It must be left to future observations to determine positively whether, in primary sclerosis of the lateral columns, the innermost divisions of the anterior columns, which, according to Flechsig, are likewise to be reckoned as part of the pyramidal tracts, are also diseased.

The degeneration may extend over the greater portion of the length of the cord, from the lumbar region up into the medulla oblongata, and even beyond that, showing a somewhat different configuration and intensity of the process in the different transverse sections.

Microscopically we encounter the ordinary picture of gray degeneration. In view of the incompleteness of the investigations thus far made, it is not yet possible to establish a positive histological distinction between primary lateral sclerosis and secondary degeneration of the lateral columns.

Among the possible *complications* of lateral sclerosis, we must mention sclerosis of the posterior columns (a combination which was seen repeatedly by Westphal, and is also very common in the later stages of tabes); furthermore, a more or less diffuse sclerosis of the anterior columns; and, finally, a lesion of the anterior gray horns, which constitutes the rule in that form of disease described under the name of sclérose latérale amyotrophique. This degeneration of the anterior gray horns establishes itself by preference in the cervical enlargement of the cord, where it involves greater or less areas, and, in addition to the interstitial changes, especially induces degeneration, atrophy, and disappearance of the large ganglion-cells in the anterior horns.

All these statements await further anatomical investigations, to render them more precise.

Pathology of Spasmodic Spinal Paralysis

Symptoms.

The *general picture of the disease* is that of motor paraplegia, generally developing itself very slowly, and gradually advancing upwards, to which symptoms of motor irritation early ally themselves—muscular twitchings, muscular tension, rigidity, and contractures, which lend to the disease-picture a peculiarly fertile character. To this is added a very striking increase of the reflex activity of the tendons, much more rarely also of the reflex action of the skin, while there is an entire absence, at least for a long time, of any serious disturbances of sensibility, of the vesical or sexual functions, and of nutrition, and cephalic symptoms likewise never arise.

The disease often begins at once, without any further precursors, with the manifestations of *motor weakness* in one or both lower extremities.

In other, not rare cases, however, this is preceded, for a longer or shorter time, by evidences of *sensitive irritation*—pain in the back, dragging and tearing pains in the legs, formication and other paræsthesias, etc. But these are rarely severe, and are generally of a transitory nature and easily subdued.

The motor disturbance, which sometimes begins in both lower extremities at once, sometimes in one alone, and very rarely in the upper extremities, consists at first only in *weakness*, in being *more easily fatigued*, and in a certain *heaviness of the limbs.* This gradually becomes actual *paresis;* the legs grow quite heavy, the gait becomes dragging, difficult, unsteady. Only in rare cases, and usually not until the later stages, does this paresis increase to complete *paralysis.* No ataxy appears during the entire course.

But distinct *manifestations of motor irritation* early ally themselves to the paretic symptoms. At first single lighter *twitchings*, or jerking of the legs, arise, which occur when sitting or lying down, often appear at night, and are very frequent when fatigued; or there may be a certain *spasmodic stiffness*, which arises on the execution of certain movements and lasts for a few moments. This gradually increases to more continuous stiffness, to regular *tension of the muscles*, which appears on active, and still more on passive motion, and affects the patient's gait; and finally it comes to permanent and severe *contractures*, which fix the limbs in a position of extension.

From all this, as long as the patients are still able to walk, there results a very characteristic method of walking, the *spastic gait*, which we have already previously described in brief (p. 97). At first but slight indications of it are to be observed; later the disturbance grows more distinct; the dragging and trailing of the legs, the drooping of the toes on the ground, the shuffling, the disposition to raise oneself on the toes, keep growing more marked, and in especially well-pronounced cases a hopping sort of raising of the body takes place with each step, and a tendency to fall over forward.

On merely placing the point of the foot on the floor, while sitting, a tremor very generally sets in, evidently in no respect different from the clonic trembling on passive dorsal flexion of the foot. And this is caused by the ever-present *high degree of increase in the reflex action of the tendons*, which can generally be demonstrated very early. Not only does the normal reflex action of tendons show a very abnormal increase, but this also appears quite actively in many tendons which under normal relations show no trace thereof.

Objective examination reveals *no disturbance of sensibility.* The sensibility of the skin and muscles, tested with the greatest care and in all directions, appears entirely normal. Not the slightest swaying appears on closing the eyes, nor any trace of unsteadiness in the twilight. In part of the cases the reflex action of the skin appears increased; generally, however, it is normal, sometimes rather diminished. There is *no atrophy of the muscles. The functions of the bladder and rectum are entirely normal,* as are also the *sexual functions.* But little is generally to be seen of vaso-motor disturbances, neither are there bed-sores nor any other trophic disturbances of the skin. The functions of the brain and the cranial nerves are entirely undisturbed.

During the further progress of the disease *the disturbances gradually continue to advance upward.* The muscles of the back and abdomen become paretic and rigid, the movements of the trunk grow difficult, sitting up and holding oneself erect become difficult or quite impossible.

Then the affection extends to the arms; this often occurs tolerably early, but generally not till the later stages of the disease. Paresis, heaviness, and stiffness of the arms sets in; examination shows here also an increase in the reflex action of tendons, but no disturbance of sensibility, no ataxy; finally, rigidity and contractures also occur, but rarely full paralysis.

This method of development is not the only and invariable one. Sometimes the affection passes from one lower extremity first to the upper extremity of the same side, and the affection may persist for a long time (even for several years) in *this hemiplegic form* before the other lower extremity is attacked. Sometimes, too, the disease begins in the upper extremities, and, *running its course downward,* does not involve the lower extremities until later.

When the disease has reached a certain point of development, it may then remain stationary for a variable length of time, often for many years, or show but a scarcely perceptible advance.

Generally, however, the paralysis eventually becomes complete, the contractures increase in intensity, and the patients, grown stiff and immovable, are doomed to permanent lying still. At the same time, there may be an entire absence of pain and of other disturbances of sensibility, the vegetative functions may take their regular course.

The disease in itself does not appear directly to threaten life, and patients generally die of some intercurrent disease.

Now and then, however, death is doubtless induced by the extension of the processes within the spinal cord to structures of the same, which are more important to life. The complications of the disease—always rare—which betray such an extension of the same, are muscular atrophy, bulbar symptoms, disturbances of sensibility, vesical weakness, cystitis, bed-sores, etc.

In estimating the importance of individual symptoms, we must principally direct our attention to the paresis and paralysis, the spasms, rigidity, and contractions, to the peculiarities of the gait resulting therefrom, and to the heightened reflex action of tendons.

The *manifestations of motor paralysis* generally begin with some *fatigue*, *insecurity*, *weakness*, and *stiffness* of the lower extremities, sometimes of both at once, but not rarely of one leg somewhat earlier than the other.

The legs feel as heavy as lead; every prolonged exertion fatigues the patients extremely; finally, longer marches and walks become impossible; the gait grows distinctly dragging.

Gradually it comes to *distinct paresis;* patients are no longer able to raise themselves on their toes, much less to maintain themselves thereon; it becomes difficult or impossible for themto raise their legs, if extended, while lying down; the leg, extended at the knee, offers less resistance to attempts at passive motion.

At the same time all movements are quiet and uniform, only sometimes rather uncertain on account of the paresis. No sign of ataxy is to be noticed. All movements called for can be executed just as quietly and certainly with closed as with open eyes.

Finally, complete paralysis may be developed; it is generally confined to the domain of single nerves and muscles, but may gradually extend further. The paralysis seldom becomes quite complete; that entire immobility of the limbs, so characteristic of many cases of myelitis, does not readily occur here.

This is the usual picture. But sometimes it happens that the disease begins unilaterally, and retains its *hemiplegic form* for quite a while, a point to which Berger called attention in his first work; the paresis then extends from one leg first to the arm of the same side, and may exist in this way for a long time (as much as several years) before the weakness invades the other leg, and finally the other arm. In all these cases the leg is more paralyzed than the arm.

But now and then the affection also begins in one or both upper extremities, and the legs are not invaded by the paralysis until later. In these cases, however, the paralysis of the legs is liable soon to become quite as intense as that of the arms.

Very soon after the signs of paralysis, occasionally almost simultaneously with these, *manifestations of motor irritation* also show themselves.

At first they are merely *single jerkings*, which arise under the most varied circumstances while sitting, lying in bed, etc., and often appear to the patient like a sort of starting from fright. The foot is suddenly raised, or the leg thrown somewhat into the air, or, on making voluntary movements, a species of cramp sets in, single muscles or the entire leg growing temporarily stiff. Often the familiar *trembling* on setting down the toe of the foot occurs, especially if the patients are somewhat fatigued.

Then distinct *muscular tension* is developed. At first, on performing passive motion, there is a doughy, gradually increasing resistance, which can be overcome by increasing pressure and somewhat lessened by repeated movements, but which again appears distinctly, especially on brisk attempts at movement. Soon the tension also shows itself in active movements, renders them difficult, makes them uncertain, and thus causes the paresis to appear greater than it is in reality.

Finally it comes to a permanent greater *rigidity of the muscles*, which gradually goes on developing to a regular and high degree of *permanent contracture.* This generally appears in the lower extremities in the form of a contracture with extension. The legs are held in the position of rigid extension, the feet in the extreme position of talipes varo-equinus, the toes sometimes flexed on the dorsum, and furthermore the legs are rigidly held together by the extreme contracture of their adductors. The rigid immobility of the legs is now and then interrupted by a clonic trembling of the feet, which may extend to the entire legs, which apparently often arises spontaneously, but is probably to be referred to a dorsal flexion of the foot produced in a reflex manner or called forth by touching the point of the foot, and which may probably now and then also be referred to a voluntary effort. Contractures with flexion rarely occur in the lower extremities, and then only in the latest stages.

The upper extremities are visited with contractures much less frequently and intensely; the arm is then drawn to the trunk, the forearm is in half flexion and in pronation, wrist and fingers in a high degree of flexion. All these contractures naturally increase the helplessness of the patient, and are a greater hindrance to the use of the limbs than the paresis.

As long as patients can still walk, these paralytic and spasmodic manifestations result in an extremely peculiar and thoroughly *characteristic gait*, which we have already designated as the "spastic gait" (p. 97).

The first thing noticeable is a certain dragging of the limbs; the feet are drawn forward with difficulty; they seem to cling to the ground, and, as they slide forward, produce a characteristic scraping noise; the tip of the foot catches on every little inequality of the ground, and patients readily stumble and fall. The gait is thereby rendered uncertain and swaying; at every step the patient changes his centre of gravity from one side to the other. The shoes mainly wear off at the toes and outer border.

In the higher grades of the disturbance, patients show a distinct tendency to raise themselves on their toes; the friction that takes place between the ground and the feet causes a certain disturbance of equilibrium in the upper portion of the body which is tending forwards, and thus the disposition arises to fall over forwards and in walking to adopt an increasingly rapid step. In some instances this is further associated with a peculiar hopping movement, a raising oneself up on the toes with every step, which produces a very remarkable effect.

At the same time the legs are carried in a stiff and constrained manner; they are locked closely to one another and somewhat sunken at the knees; the steps are small and hesitating.

It is evident at the first glance how sharply this variety of gait is distinguished from the ataxic. There is no trace of the flinging motion of the legs, of throwing forward or turning outward the tip of the foot, of stamping the heels, of holding the knee stiff to a degree bordering on hyper-extension, as in ataxy; we see, rather, the opposite of all this.

The peculiarities of the spastic gait are very easily explained —in part by the paresis, which causes the dragging and the catching of the toes; in part by the muscular tension, which brings about the stiffness of the movements, the closed position of the legs, the short steps, the scraping with the tip of the foot, the walking on the toes; and in part by the heightened reflex action of the tendons, which probably explains the hopping movement with every step.

At last, of course, all walking ceases, and patients are able, at most, to drag themselves around on crutches, or, with the help of their hands, to move about by holding on to chairs and furniture, dragging their legs after them like two sticks, and only now and then using them as props. Finally, patients are doomed to continuous lying in bed, as sitting up often becomes downright impossible, on account of the stiffness of their legs.

We have already, at various places in this work, referred to another important, and, as it would appear, absolutely constant symptom of the disease—the *great increase in the reflex action of tendons.* This may be observed to a very intense degree in spasmodic spinal paralysis, inasmuch as not only those tendon-reflexes which normally exist in the majority of people show a greatly increased intensity, but many other tendons likewise (and even other aponeurotic structures) may be excited to active reflex twitching through mechanical irritation.

The *reflex action of the tendon of the patella* is most constantly increased—not rarely to such an extent that it can be maintained in a clonic form by the sudden, forcible fixation of the patella with a blow downwards. Not less constant is the increase in the *reflex action of the tendo Achillis*, which can generally be maintained in the clonic form on passive dorsal flexion of the foot. The *reflex activity of the tendons of the adductors* generally behaves in the same way. I have repeatedly been able to excite this by tapping over the region of the lumbar vertebræ. Tendon-reflexes, furthermore, occur in the lower extremities in the tibialis anticus and posticus, in the biceps femoris, the semi-tendinosus, etc. Reflexes in the quadriceps also sometimes originate in the upper broad end of the tibia.

The reflex action of the tendons is generally no less well developed in the *upper extremities.* It is to be found in the biceps and triceps humeri. Quite remarkable reflexes can be aroused from the lower end of the radius, as well as from the ulna, by tapping—the first in the biceps, the latter in the triceps and the posterior portion of the deltoid. The flexors of the fingers and the extensors of the wrist, as well as the supinator longus, can be excited in a reflex manner by their tendons; also sometimes the interossei by tapping on the metacarpal bones, the deltoid from the spine of the scapula, the pectoralis major from the sternum, etc.

It hardly requires demonstration that in all these cases we have to deal with nothing else than a *heightening of the reflex processes.* This appears as an absolute necessity, from some of the facts heretofore communicated, entirely aside from the circumstance that all authors who, since the appearance of Westphal's works and my own, have occupied themselves particularly with this circumstance, have come to the conclusion that the physiological phenomena mentioned could only represent reflex processes. We must therefore refer the increase in the tendon-reflexes which arise in spasmodic spinal paralysis to an increase in the reflex activity of the spinal cord.

The singular part of it is that this increase is almost always confined to this one class of reflexes ; for the skin reflexes by no means constantly show any similar relations. I found them distinctly increased in hardly one-third of my cases ; in the rest generally normal—often not at all distinctly present, but also often tolerably lively, even if not exactly abnormally heightened. At all events, however, the reflex activity of the skin by no means bore any relation to that of the tendons, and the increase of the former certainly does not constitute an essential symptom of spasmodic spinal paralysis.

The *electrical excitability of the motor nerves* never shows any qualitative, but always merely insignificant quantitative changes; in all the cases more carefully examined, I found a distinct, though slight, *lowering of the faradic and galvanic excitability.* I have never yet found an increase of the same.

All the other symptoms that remain to be noticed are of an essentially negative quality ; but this very fact contributes not a little to characterize the disease. The entire *absence of every disturbance of sensibility*, even on the most careful testing of the sensibility of the skin and muscles; the *absence of vesical and sexual weakness;* the *absence of muscular atrophy and of bed-sores;* the entire *absence of disturbances of the brain and cranial nerves*,—are at least as significant and as important for the characterization of the disease as the presence of paresis, muscular tension, and increased reflex action of tendons.

Other isolated symptoms—and indeed of the most varied kind—do occasionally arise, but appear as more or less accidental features in the disease, and will be more appropriately discussed under the head of Complications.

Course, Duration, Terminations.

The *course* of the disease, in the majority of cases, is very slow, insinuating, and chronic. Months and years may pass before the manifestations have so far developed that the disease can be recognized with certainty. And then the trouble often remains stationary for years. In some cases, however, the manifestations develop more rapidly, and may within a few months assume the form of the typical disease-picture.

The *duration* of the disease is always of many years, and is generally to be reckoned by decades. Complicated cases only—of which we shall speak below—are sometimes of short duration, and run their course to a fatal termination within a few years.

Among the *terminations* of the disease, that in recovery is probably the rarest, although not quite as uncommon in this form as in other forms of chronic spinal paralysis. Already in two cases I have seen complete or almost complete recovery set in. Several cases have been materially improved.

Generally, however, the disease remains stationary for an indefinite length of time, or makes extraordinarily slow progress. It does not seem, of itself, to threaten life directly; in most cases death occurs through accidental, intercurrent diseases.

If complications arise, these may cause death; thus, for instance, bulbar symptoms, or the manifestations of severe spinal paralysis, such as cystitis, bed-sores, etc., with their evil consequences, which produce gradual exhaustion of the patient.

Theory of the Disease.

The theory of spasmodic spinal paralysis cannot as yet be firmly established, on account of the lack of reports of post-mortems. But some isolated conclusions may still be drawn from our present physiological and pathological knowledge, and these will here be given in brief.

The essential features in the symptom-picture of spasmodic spinal paralysis are, on the one hand, the *presence* of paresis and paralysis, muscular tension and contractures, and the heightening of some kinds of reflex action (that of the tendons), and, on the other hand, the *absence* of disturbances of sensibility, of vesical and sexual weakness, of trophic disturbances of the skin and muscles, of ataxy, and of all brain symptoms.

From these latter negative symptoms we may, with considerable certainty, exclude certain divisions of the spinal cord from participation in the disease, viz.: the posterior columns, the posterior gray horns, the central gray substance, and probably also a great part of the anterior gray horns; of course, also the brain.

We therefore only have the so-called motor portions of the spinal cord left for the localization of the disease, that is, the antero-lateral columns, and perhaps also a portion of the gray substance.

More recent physiological investigations unanimously indicate that the lateral columns at least quite certainly contain motor tracts, while we are still uncertain with regard to the functions of the greater part of the anterior columns proper. An irritation and paralysis of these motor tracts in the lateral columns, caused perhaps by an inflammatory affection, might accordingly very well explain the muscular tensions and pareses present in our disease. The entire absence of atrophy probably justifies us in excluding, with some certainty, disease of the motor tracts within the gray substance; the same evidence, as well as the failure of the reflexes, positively excludes disease of the anterior roots—therefore probably also disease of those portions of the anterior columns traversed by these roots. If, on the basis of our present physiological knowledge, we are to refer the existing manifestations of motor irritation and paralysis without atrophy of the muscles to any lesion of the spinal cord, we are thus pointed to the lateral columns, especially to the posterior divisions of the same, which contain the so-called pyramidal tracts of the lateral columns [Pyramidenseitenstrangbahnen] (Flechsig).

Then the increase of reflex action would still remain to be explained. This can hardly be referred to disease or increased irritability of the gray substance, as all other symptoms of disease of the gray substance are wanting. The only explanation then lies in a lesion of the tracts which control reflex action; according to the investigations of Woroschiloff, these also lie in part within the lateral columns; consequently we may with propriety refer the symptom of increased reflex action of the tendons also to a disease of the lateral columns.

Pathological experiences coincide with this in a significant manner. If a unilateral paralysis, caused by cerebral apoplexy, has contractures and increased reflex action of tendons associated with it, anatomical investigation uniformly demonstrates the familiar signs of descending secondary degeneration of the lateral columns of that side. In the numerous affections of the spinal cord, which present as one of their manifestations secondary degeneration of the lateral columns (transverse myelitis, myelitis from compression, hæmatomyelia, multiple sclerosis, etc.), muscular tension, contractures, and increased reflex action of the tendons, are found in addition to the symptoms of the fundamental affection. In amyotrophic lateral sclerosis Charcot has several times anatomically demonstrated sclerosis of the lateral columns as a very probable cause of paralysis, contractures, and increased reflex action of the tendons. In a case of combined sclerosis of the posterior columns and the lateral columns, Westphal,[1] in addition to disturbances of sensibility, found paralysis with muscular twitchings and muscular tension, and increased reflex action of tendons. As these symptoms never occur in consequence of sclerosis of the posterior columns, they cannot well be referred to anything else than the sclerosis of the lateral columns.

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[1] Archiv f. Psych. u. Nervenkrankh. Bd. V. p. 822. 1875.

Physiological and pathological experiences, therefore, with a unanimity that is worthy of consideration, point to the lateral columns as the seat of disease; and we shall probably not go amiss if, as Charcot did long ago, we accept a *chronic inflammatory process, a sclerosis of the posterior divisions of the lateral columns, as the highly probable anatomical basis of spasmodic spinal paralysis.*

But, unfortunately, we thus far lack conclusive reports of post-mortems. Until we have such, this assumption remains only a very probable one; and, in view of the exceedingly vacillating character which still attaches to our "physiological and pathological experiences" concerning the spinal cord, we shall do well to await the definitive confirmation of this view before exchanging our clinical designation for an anatomical one.

Complications.

The disease-picture of spasmodic spinal paralysis does not always appear in its clear, typical form. Quite apart from those cases in which this constitutes only one of the features in the symptom-picture of other diseases, and is probably to be regarded as the expression of the secondary degeneration of the lateral columns, we also encounter in this form of disease, as well as in numerous other spinal diseases, single cases in which, while the symptom-picture of spasmodic spinal paralysis decidedly dominates, it still appears complicated with single symptoms or with whole series of symptoms, which give evidence of the extension of the disease to neighboring or remote portions of the spinal cord.

In my detailed work alluded to above, I have touched somewhat more specifically on these relations, and have pointed out the fact that there are two series of symptoms in particular which complicate the disease-picture of spasmodic spinal paralysis in this way, and may render the diagnosis more difficult. On the one hand there are the *symptoms of sclerosis of the posterior columns*, of actual tabes (slight disturbances of sensibility, swaying on closing the eyes, slight vesical weakness, lancinating pains, distinct ataxy, etc.); on the other hand, the symptoms of a more or less extensive, more or less progressive *atrophy of the muscles*, which arises by preference in the upper extremities.

So far as complicated cases of the first variety are concerned, to which Berger already drew attention, all possible gradations of tabes symptoms occasionally take place in them, sometimes only slight subjective disturbances of sensibility, combined with some vesical weakness, sometimes lancinating pains, paræsthesias, and the sense of a tight girdle, combined with indications of ataxy, swaying on closing the eyes, etc.; sometimes the tabes symptoms (anæsthesia, ataxy, vesical and sexual weakness) may even stand in the foreground, and only the simultaneous presence of paresis, muscular tension, and especially of increased reflex action of tendons, gives evidence of the existence of spasmodic spinal paralysis likewise. There are also cases in which the diametrically opposite of the typical forms of spasmodic spinal paralysis and tabes are more or less mingled, and in which one may be in doubt whether to credit them more to the one or to the other form of disease.

The doubt in such cases may be most easily solved on the assumption—which seems the most natural and the most applicable—that we here have to deal with a combination of sclerosis of the posterior columns with sclerosis of the lateral columns. Such cases have frequently been seen—their existence in the last stages of tabes is well enough known,—and Westphal, in the work cited above, especially pointed out their occurrence. But this question, too, can only be cleared up by conclusive results of post-mortem examinations.

In the complicated cases of the second variety, likewise, there are *all possible gradations of muscular atrophy* to be observed. Sometimes one sees merely a simple, not very high grade of emaciation of the paralyzed extremities, with or without scanty fibrillar contractions, or there are more distinct atrophic conditions, which may remind one of beginning progressive muscular atrophy. This is seen particularly in the upper extremities, and is sufficiently designated by the atrophy of the small muscles of the hand, the sinking of the inter-metacarpal spaces, the characteristic claw-shape of the hands, etc. But recently two cases have fallen under my observation in which the typical manifestations of spasmodic spinal paralysis were supplemented by this atrophy of the muscles of the hand on paretic upper extremities.

To explain this complication, we must suppose that the sclerotic process, which we believe to exist in the spinal cord, extends to the gray anterior horn—with special ease and frequency in the cervical enlargement—and from here produces the muscular atrophy by lesion of the large ganglion-cells.

But the highest degree of this combination of spasmodic spinal paralysis with muscular atrophy is presented by those cases in which the atrophy of the upper extremities shows a rapidly progressive character, in which bulbar symptoms associate themselves with it sooner or later, and within a few years the disease leads to a fatal termination.

These cases, which were formerly generally counted in with actual "progressive muscular atrophy," but which may be sharply distinguished from the typical form of the same, have of late been more carefully investigated, anatomically and clinically, by Charcot, who has drawn a disease-picture of the same in his customary masterly manner, and given it the name of

Sclérose Latérale Amyotrophique.

We will here devote a little space to the discussion of this disease, as it appears to us to bear an undoubted relationship to spasmodic spinal paralysis, although it differs from the latter in some not unimportant points. As we do this in a manner that implies no prejudgment of the case, we reserve the right, in case further investigation of this interesting disease should justify it, to assign to it another more appropriate place.

According to Charcot, amyotrophic lateral sclerosis is characterized as follows:

The disease *generally begins in the upper extremities* with *motor weakness*, to which a more *diffuse muscular atrophy* is soon added, with fibrillar twitchings, etc. Then *muscular tension* and *contractures* are developed, which bring the arms into a permanently deformed position: the upper arm is pressed fast to the trunk, the forearm half flexed and pronated, hand and fingers strongly flexed. Whatever movements can still be executed are weak and tremulous. The muscular atrophy progresses rapidly; sometimes there is lipomatous hypertrophy of the same.

In a case, evidently belonging here, and which is furthermore of interest, because the disease is developed on a young man who carries about him the sequels of an attack of the "spinal paralysis of childhood," galvanic examination shows the most pronounced reaction of degeneration in the extremely atrophied muscles of the upper extremities, while the excitability of the nerve-trunks is nevertheless preserved. In a very marked case, Berger found normal qualitative and quantitative electrical excitability in the nerve-trunks.

After a longer or shorter time, usually after from six to nine months, *the lower extremities are also attacked;* sometimes formication and numbness set in, always paresis, advancing to final paralysis; to these are added muscular tension, permanent contractures, generally contractures in the position of extension, increased reflex action of tendons. At first there is no atrophy in the lower extremities; the muscles are tense and firm; no anæsthesia, no paralysis of the sphincters, no bed-sores. It is not till very late that fibrillar contractions take place, and gradually diffuse atrophy of the legs; then the contractures diminish.

Finally, in a third stage, the manifestations of *bulbar paralysis* are added to the preceding: paralysis and atrophy of the tongue and lips, paresis of the pharynx and larynx, finally disturbances of respiration, which before very long induce death.

The malady develops itself rapidly, and is said always to induce death in from one to three years.

The characteristics of the disease then are: the paralysis and contracture, with rapid, diffuse muscular atrophy in the upper extremities; the paralysis, with contracture and afterwards atrophy in the lower extremities; the final manifestations of bulbar paralysis.

In a number of post-mortems, Charcot, Joffroy, Gombault, and others, found symmetrical sclerosis of the lateral columns and degenerative atrophy of the anterior gray horns, with loss of the large ganglion-cells.

Charcot refers the paresis and contracture to the sclerosis of the lateral columns; the muscular atrophy to the degeneration of the gray substance; the bulbar paralysis to the extension of this degeneration to the nerve-nuclei in the medulla oblongata.

The lateral sclerosis is said to be the primary step; from the lateral columns the process passes over to the anterior gray horns, perhaps by conduction along the physiological tracts which lead from the lateral columns to the large ganglion-cells of the anterior horns.

The distinction between amyotrophic lateral sclerosis and the typical form of spasmodic spinal paralysis is evident from the earlier involvement of the upper extremities, from the supervention of muscular atrophy and of bulbar paralysis, and, finally, from the rapid course of the former. It differs from progressive muscular atrophy in the fact that, in amyotrophic lateral sclerosis, the atrophy is preceded by paralysis, and that there are contractures in the uniform atrophy which extends over whole muscular masses, and in its decidedly more rapid pernicious course.

According to the experiences thus far before us, which are of course by no means final, the prognosis of amyotrophic lateral sclerosis seems to be absolutely unfavorable.

Further investigations with regard to this interesting form of disease are greatly to be desired.

Diagnosis.

The disease-picture of spasmodic spinal paralysis is so characteristic that it can always be recognized with the greatest ease, especially when it is uncomplicated. The sum of the positive symptoms (paresis, paralysis, muscular tension, contractures, increased reflex action of tendons, a development which is slowly and, as a rule, systematically progressive from below upwards, etc.), and of the negative symptoms (absence of anæsthesia, of vesical and sexual weakness, of atrophy, of bed-sores, of cerebral symptoms), usually establish the prognosis with certainty.

We must remember to distinguish it from the following forms of disease:

First, from *myelitis transversa* (myelitis from compression, hæmatomyelia, etc.). In this we regularly find, besides the motor, also sensitive paralysis of various degrees, vesical weakness, bed-sores; the paraplegia is developed more rapidly and completely, but the upper boundary of the paralytic manifestations remains stationary. Reflex action of the skin, as a rule, is materially heightened. To this are usually added extreme evidences of sensitive irritation, at the beginning; furthermore, those manifestations of the disease which indicate the existence of compression, etc.

In fairly well-marked cases, spasmodic spinal paralysis is very easily to be distinguished from *tabes dorsalis.* In tabes there are lancinating pains, the sense of a tight girdle, sensitive disturbances, the lowering of muscular sense, swaying on closing the eyes, disturbance of certain cranial nerves, ataxy, a flinging, stamping gait, vesical and sexual weakness, no paresis or paralysis, no muscular tension and contractures, the entire absence of all tendon reflexes. In spasmodic spinal paralysis we have the opposite of all this. In all typical cases, therefore, the distinction will be easily made. In those cases, however, in which a combination of the two classes of symptoms is recognizable, we shall be enabled, according to the hints given above, to recognize the greater or less participation on the part of each disease by the preponderance of one or the other set of symptoms.

This disease is to be distinguished with certainty from *poliomyelitis anterior chronica* (chronic atrophic spinal paralysis; see farther on) by the fact that, in the latter, a high degree of atrophy of the paralyzed muscles is very rapidly developed, with loss of faradic excitability and with the reaction of degeneration; that there is an entire loss of the reflex action of tendons; and that contractures, if they do occur, at first only attack the antagonists of the paralyzed muscles. The development of the paralysis is furthermore very much more rapid.

In most cases the distinction between this disease and *multiple sclerosis* is easy, except when the latter, at the beginning, makes its first and only localization in the lateral columns, and then presents itself quite under the typical picture of spasmodic spinal paralysis. In that case a diagnostic distinction between the two is simply impossible. Not until the moment when the multiple sclerosis makes further localizations, and causes these to be noticed by further more or less characteristic symptoms (sensitive disturbances, psychical changes, disturbances of certain cranial nerves, nystagmus, ataxy, trembling, disturbances of speech, etc.), shall we be able to settle the diagnosis, with increasing certainty, in favor of multiple sclerosis.

In distinguishing between *paralysis from lesions of the cauda equina* and *other peripheral paralyses*, the following may be considered as decisive symptoms: the absence of disturbances of sensibility and muscular atrophy, the retention of electrical excitability and the increase of the reflex action of tendons.

The proper significance of those cases of spasmodic spinal paralysis which assume a *hemiplegic form* will easily be recognized on giving them a little attention. They are readily to be distinguished from Brown-Séquard's unilateral lesion of the spinal cord, which we shall describe in the following section, by the absence of crossed anæsthesia and other symptoms to be described in that section. Over against *cerebral hemiplegia* we must remember that spasmodic spinal paralysis, as a rule, begins in the lower extremities, and advances but very slowly to the upper; that the upper extremities are usually much more lightly attacked; that muscular tension and contractures are developed early; that disturbances of sensibility, as well as all symptoms on the part of the head and cranial nerves, are entirely wanting. In view of these signs, it will not be easy to be mistaken.

The presence of the symptom-picture of spasmodic spinal paralysis in mixed and complicated forms of disease (transverse myelitis, myelitis from compression, multiple sclerosis, amyotrophic lateral sclerosis in connection with sclerosis of the posterior columns, etc.) will generally be easily recognized by its characteristic signs.

Prognosis.

According to the experience thus far before us, spasmodic spinal paralysis seems to run a less pernicious course than the forms of chronic spinal disease thus far studied. On the one hand, it seems to be compatible with an unlimited duration of life (if very decided complications do not exist), and this doubtless depends on the absence of those disturbances of the bladder

—cystitis, bed-sores, etc.—which so often bring a fatal termination. On the other hand, the disease is capable of improvement, and, in a few cases, also of recovery. I have thus far seen as good as complete recovery in two cases, and material improvement in three; though it is true that most cases receive no benefit from treatment. The prognosis, as regards life, is therefore, in general, very favorable; as regards recovery, it is at least more favorable than in the other forms of chronic myelitis.

If complete paralysis and extreme contractures have once set in, we can hardly expect any improvement.

Certain complications, especially muscular atrophy, bulbar symptoms, etc., of course vitiate the prognosis to a greater or less degree. Furthermore, this is naturally also dependent on the general and special circumstances of the individual case.

Therapeutics.

All that we have said above with regard to the treatment of chronic myelitis, and that of tabes, might also be applied to the therapeutics of spasmodic spinal paralysis, as this disease is doubtless most intimately related to those just mentioned.

As one of the means which has thus far, in my own hands and those of others, afforded the best results, may be mentioned *the galvanic current;* its application is to be conducted entirely according to the general principles repeatedly laid down.

Aside from this, the use of a *reasonably conducted cold-water cure* deserves the greatest confidence; gaseous *saline thermal springs* may also be used; simple thermal waters, unless most carefully managed, are objectionable.

Internally we may first try the nitrate of silver, and then the other remedies mentioned above. Strychnine is, under all circumstances, contraindicated.

With reference to diet, method of life, exercise, living in the open air, the regulating of sleep, of sexual intercourse, etc., I refer my reader to the discussion of these points as cited above.

14. *Unilateral Lesion of the Spinal Cord—Brown-Séquard's Spinal Paralysis—Hemiplegia et Hemiparaplegia Spinalis.*

Brown-Séquard, Course of Lectures on the Physiol. and Pathol. of the Central Nerv. Syst. Philad. 1860.—Recherches sur la transmission des impressions de tact, de chatouillement, de douleur, etc., dans la moëlle ép. Journ. d. l. Physiol. de l'homme et des animaux. VI. pp. 124, 232, 581. 1863. (Also contains most of the older cases.)—On spinal hemiplegia. Lancet. 1868. Vol. II. pp. 593, 659, 755, 821.—Recherches sur le trajet des diverses espèces de conducteurs d'impressions sensit. dans la moëlle ép. Arch. de Physiol. I. p. 610 u. 716. II. p. 236 u. 693. 1868 u. 1869.—*Viguès*, Plaie de la moëlle ép. dans la région dorsale. Monit. des hôp. 1855. p. 838.—*Bland Radcliffe*, Lancet. 1865. May 27. —*Basire*, Lancet. 1865. July. p. 116.—*Uspensky*, Zur Pathologie des R.-M. Virch. Arch. Bd. 35. S. 301. 1866.—*Jaccoud*, Leçons de clin. méd. Paris, 1867. p. 451.—*Perroud*, Journ. de médec. de Lyon. Vol. X. p. 385. 1868.—*Charcot*, Hémiparaplégie déterminée par une tumeur, etc. Arch. de Physiol. II. p. 291. 1869.—*C. J. F. Richter*, Fall von einseitiger Rückenmarksverletzung. Diss. Berlin, 1868.—*M. Rosenthal*, Ueber spinale Halbseitenlähmungen. Oesterr. Zeitschr. f. pr. Heilk. 1867. Nr. 47–52.—Klin. d. Nervenkrankh. 2. Aufl. S. 395. 1875.—*Puoluzzi* (*Cantani*), Affez. di una meta laterale del midoll. spin. etc. Morgagni. Disp. XI. 1870.—*Fieber*, Klin. Stud. üb. d. Brown-Séquard'sche Spinallähmung. Wien. med. Zeitung. 1871. Nr. 21–23.—*W. Mueller*, Beitr. zur pathol. Anat. und Phys. des R.-M. Fall 1. Leipzig, 1871.—*Joffroy et Solmon*, Plaie d. l. moëlle ép. dans. la rég. dorsale. Gaz. méd. de Par. 1872. Nr. 6–8.—*Charcot* et *Gombault*, Lés. dissém. des centres nerv. chez une femme syphil. Arch. d. Phys. V. p. 143. 1873.—*Riegel*, Halbseitige Rückenmarksverletzung. Berl. klin. Woch. 1873. Nr. 18.—*Troisier*, Deux cas de lésions scléreuses, etc. Obs. II. Arch. d. Physiol. V. p. 709. 1873.—*Lanzoni*, Sifiloma sulla porzione infer. etc. Il Morgagni, 1874. Marzo.—*M. Bernhardt*, Halbseitige Rückenmarksverletzung. Arch. f. Psych. u. Nervenkrankh. IV. S. 227. 1874.—*Vix*, Einseitige Verletzung des verl. Marks u. s. w. Correspondenzbl. d. ärztl. Ver. im Rheinland. 1874. Nr. 14. (Centralbl. 1875. Nr. 22.)—*Burresi*, Lo Sperimentale. 1871. Gennajo. 1875. Nov. (s. Virchow-Hirsch Jahresber. 1871 u. 1875.)—*G. v. Dall'Armi*, Halbseitige Verletzung des R.-M. Diss. Würzb. 1875.—*Feder. Alessandrini*, Ferita della meta destra del mid. cervic. infer. Annal. univers. Oct. 1876.—*H. Koebner*, Die Lehre von der spinalen Hemiplegie. Deutsches Arch. f. klin. Medic. Bd. XIX. S. 169. 1877.

Introduction and History.

This section treats, not of a single, definite form of disease, but of various different diseases of the spinal cord (such as traumatic injuries, inflammation, compression, sclerosis, tumors, syphilis, etc.), all of which, however, by reason of their definite localization, acquire such characteristic features that they may be treated of under one common head. The very peculiar picture of disease here presented depends on *the localization of the trouble in one lateral half of the spinal cord*—the more or less complete destruction of one entire lateral half of a transverse section of the cord, while the longitudinal extent of the diseased process may vary exceedingly.

To Brown-Séquard, in particular, belongs the credit of having exhaustively studied this group of symptoms, both by means of clinical observations on the human subject and by numberless experiments on the lower animals, and of having established, beyond all doubt, the fact of its frequent occurrence. He was the first who asserted, with all positiveness, that the sensory fibres decussated in the spinal cord already, and who thereupon demonstrated that section of one-half of the spinal cord produced quite a peculiar group of symptoms, the chief characteristic of which consists in the crossed sensory and motor paralysis. This assertion gave rise to a long and vigorous war among physiologists, which may now be considered as virtually settled in favor of Brown-Séquard.

In his great work, which appeared in 1863, this author also endeavored to establish these physiological facts as applying to man, and for this purpose collected and critically worked up such clinical material as could be obtained. In so doing he created the pathology of unilateral lesions.

It is true that, before Brown-Séquard's labors, a number of cases belonging under this head had been described in literature (for example, by Monod, Charles Bell, Gintrac, and others), but generally without any comprehension of the pathogeny of the same. These cases were first caused to be understood through the labors of Brown-Séquard.

Since the accomplishment of this fundamental work, numerous observations have been published under this head. In some few, immaterial points, they have served to extend and render more precise the doctrines of Brown-Séquard, but in all essential points they have served only to confirm and establish those doctrines. Quite recently Koebner has put forth an ample treatise on this affection, based on a collection of most of the known cases.

Definition.

What we propose to do here is to present a brief and essentially symptomatic picture of those forms of spinal disease which, *anatomically speaking*, have only this much in common—that they are more or less sharply and more or less completely localized on one-half of the spinal cord, while they may vary as to their longitudinal extent, or as to the height at which they are situated.

These cases are *clinically* characterized by unilateral motor paralysis—either hemiparaplegia or hemiplegia—on the side of the lesion; by crossed anæsthesia on the opposite side of the body; by symptoms referable to the roots of the nerves, and corresponding exactly with the height at which the lesion is situated; and by a series of other manifestations which may vary very much, according to the seat and extent of the process, but which are also severally very characteristic of the different portions of the spinal cord which may be affected.

Etiology and Pathological Anatomy.

As we have in this connection to deal with various kinds of lesions of the spinal cord, we cannot very well speak of any special etiology. The causes which respectively give rise to these various lesions may also serve as causes of the unilateral paralysis.

It is only necessary here to enumerate those pathological processes in the spinal cord which occasionally affect one-half of the cord; if any one so chooses, he may regard these as the immediate causes of the "unilateral lesion."

By far the most frequent cause is to be found in *traumatic injuries* which happen to involve only one-half of the spinal cord, and thus, as it were, perform the experiment of cutting one lateral half of the cord in man. Instances of wounding of the spinal cord by blows with a knife or dagger are especially frequent in literature (see the cases of Brown-Séquard, Viguès, W. Mueller, Riegel, Joffroy, Vix, and others). Such pointed instruments may very easily reach the spinal cord through a wound at the side of the spinal column, may penetrate the cord more or less deeply, and more or less completely divide one-half of the same. The position of the intervertebral fissures readily explains why, in the majority of such instances, only one-half of the cord is wounded.

Compression of the spinal cord from without is the next cause which not infrequently affects one-half of the cord mainly or exclusively, thus giving rise to the manifestations of a unilateral lesion. This may arise from meningeal tumors (cases of Charcot, Gintrac, Oré), or from fracture or dislocation of the vertebræ (cases of Brown-Séquard), or doubtless, also, through intrameningeal effusion of blood. It is probably in this category that we should place those cases in which the manifestations of a unilateral lesion are developed after falling headlong or falling upon one's back.

But the same array of symptoms may likewise be called forth by processes developed within the spinal cord itself in one of its lateral halves. This has been seen to occur as the result of *hemorrhage* in one lateral half of the cord (Monod's case); as the result of a unilateral *circumscribed sclerosis* (Troisier's case); as the result of *chronic myelitis*, developed in consequence of taking cold or of concussion of the spine, or spontaneously and gradually (Uspensky, Radcliffe, Paoluzzi, etc.); as the result of an *intramedullary tumor* (Gendrin); and finally, as the result of *syphilitic affections* (Charcot et Gombault, Lanzoni). In short, all possible acute or chronic affections of the spinal cord may produce the features of unilateral lesion whenever they extend over the greater part of *one* lateral half of the cord.

Symptoms.

The characteristic group of symptoms belonging to a unilateral lesion may either be developed very gradually and insidiously or quite suddenly. This, of course, depends altogether upon the nature of the fundamental lesion: the stab of a knife calls the entire group of symptoms into being at one blow; a circumscribed sclerosis induces but a very gradual development of these symptoms.

The peculiar signs of this affection are as follows: On the side of the lesion there is *motor paralysis*, which is confined to this side, therefore is *unilateral*, which may merely involve one leg (hemiparaplegia), or, if the lesion is situated higher up, may also attack the arm of the same side (spinal hemiplegia). A marked contrast is presented by the other side, the one opposite to the seat of the lesion, which is either entirely free from all motor paralysis, or is affected only to a slight and subordinate degree.

Evidences of *vaso-motor paralysis* are also generally found *on the side of the lesion*, especially if the affection has been rapidly developed (as in traumatism, hemorrhage, etc.). The temperature of the paralyzed limbs appears more or less elevated, to the extent of 1.8° Fahrenheit and more.

Muscular sensibility and *muscular sense* are diminished on the affected side, according to the evidence of various observers (Brown-Séquard, Burresi, Vix). Lanzoni also found *electro-muscular sensibility* lowered on the affected side; while in one case, in which, it is true, the paralysis was but incomplete, Bernhardt found the so-called *sense of power* [Kraftsinn] retained.

The *sensibility of the skin*, on the other hand, acts very remarkably. The entire paralyzed side shows more or less considerable *hyperæsthesia* for all qualities of sensation. Impressions of touch, of temperature, or of pain are recognized with extraordinary and even with painful acuteness; sensitiveness to tickling is exceedingly marked and disagreeable; the test by means of Weber's compasses usually demonstrates greater acuteness in the power of localizing sensations of touch. Sometimes, however, this hyperæsthesia is limited to certain single varieties of sensibility.

The hyperæsthetic region of skin is, in many instances, bounded above by an *anæsthetic belt*, generally narrow, though sometimes wider, which corresponds exactly with the height and longitudinal extent of the lesion in the spinal cord. In some instances another *narrow hyperæsthetic zone* may be demonstrated above the anæsthetic belt, the former extending, although with less distinctness, to the opposite side, and surrounding this likewise as with a girdle.

The conditions of *reflex action* on this, the paralyzed side, are variously reported in different instances: Paoluzzi, Riegel, and others have found reflex action increased; Bazire, Brown-Séquard, and others have found it diminished. Very special interest might here be attached to the state of reflex irritability of the tendons, and yet, up to the present time, this has, to all intents and purposes, not been tested at all. In one case of unilateral lesion caused by an intramedullary tumor, I found the reflex irritability of the tendons materially increased. In a case of traumatic unilateral lesion Joffroy and Solmon report the existence—though not until several weeks after a traumatic unilateral lesion—of clonic spasm of the dorsum of the paralyzed foot. (Secondary degeneration of the lateral column ?)

On the side of the body opposite to the seat of lesion the following conditions are found:

There are *no motor disturbances*, or they are, at most, extremely insignificant; all movements can be executed easily, safely, and powerfully. The *sensitiveness of the muscles*, as well as *electro-muscular sensibility* (Lanzoni), is retained.

On the other hand, more or less *complete anæsthesia* of the skin exists to a height corresponding with that of the lesion. All varieties of sensibility—that to touch, temperature, pain, and tickling—are, as a rule, equally affected, though sometimes one may be somewhat more, another somewhat less so. There may even be very well-defined partial paralysis of sensation, as in a case reported by Koebner. The anæsthesia extends exactly to the median line of the body, as may be distinctly demonstrated on the trunk, the scrotum, etc.

Now and then a narrow, *slightly hyperæsthetic zone* may be found at the upper limit of the anæsthetic region, the former corresponding with the similar zone of the other side.

Reflex action generally seems to be retained and normal on this side, although in some cases it has been found to be increased (Brown-Séquard, Bazire). In the case above referred to I found it normal.

On this side, too, there is *no vaso-motor paralysis*—at least, in recent cases. The temperature is normal, and therefore lower than that of the paralyzed side. Sometimes, however, and this seems to be especially true where the affection has existed for a longer time, the reverse is found to be the case: the anæsthetized is warmer than the paralyzed side, which latter then shows an abnormally low temperature.

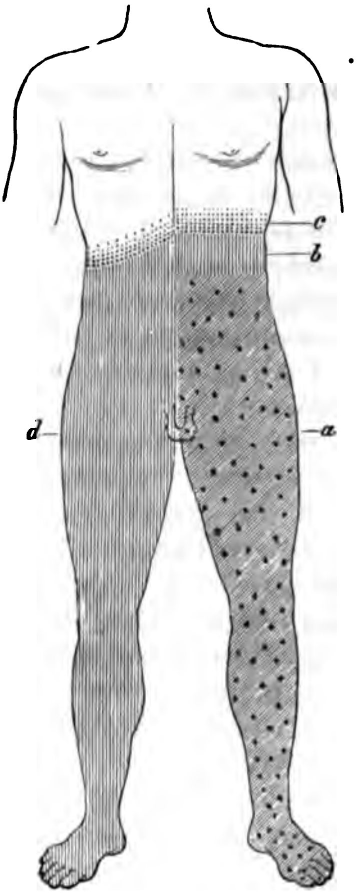

FIG. 12. Diagrammatic representation of the skin symptoms in unilateral lesion of the dorsal portion of the spinal cord on the left side. The diagonal shading signifies motor and vaso-motor paralysis; the vertical shading signifies anæsthesia of the skin; the dotted shading indicates hyperæsthesia of the skin.

In the accompanying diagram the characteristic difference in the two sides is represented, so far as it relates to the skin symptoms. It is assumed that there is a unilateral lesion of but slight longitudinal extent about the middle of the dorsal region, in the *left* half of the spinal cord. The left lower extremity and left lower half of the trunk show motor and vaso-motor paralysis, as well as hyperæsthesia of the skin (*a*); above this there is a narrow anæsthetic zone, corresponding to the longitudinal extent of the lesion (*b*); and above this again a narrow hyperæsthetic zone, which likewise extends, like a girdle, over the right side (*c*). The right side shows complete anæsthesia (*d*).

This diagram would be modified according to the seat and extent of the lesion. If the lesion is situated higher up, the upper extremity may fall within the domain of the anæsthetic zone (*b*); or, if the lesion were seated still higher, this zone would fall in the region of the throat and neck, and the upper extremity would become hyperæsthetic and paralyzed. If the longitudinal extent of the lesion increases, the width of the anæsthetic zone (*b*) also increases; it may, for instance, extend over the entire length of the trunk, and then the characteristic differences of sensibility between the two sides would appear only on the lower extremities.

Aside from these main symptoms of a unilateral lesion, other less constant and less characteristc manifestations may be observed in individual instances.

It is not uncommon to meet with a *painful feeling of constriction* at the height of the lesion; also with all sorts of *painful sensations* (burning, darting, boring pain, etc.), which sometimes occur most prominently on the anæsthetic side, sometimes on that which is paralyzed and hyperæsthetic, and sometimes on both sides.

Distinct disturbances in the evacuation of the bladder and bowels are almost always present. In the acute traumatic cases it is especially common to find severe paralytic symptoms at the beginning of the attack (complete retention or complete incontinence, involuntary stools, etc.), which, however, generally soon recede and give place to lighter, more permanent troubles (weakness of the bladder, weakness of the sphincters). Sometimes these disturbances are very insignificant.

The sexual powers act in the same way; generally are more or less diminished, though sometimes they remain unchanged.

Decided *emaciation* of the paralyzed side generally sets in quite early, sometimes making remarkably rapid progress. This symptom is also present in quite chronic cases.

Corresponding to this, the paralyzed side has, in a number of instances, shown a notable *lowering of faradic irritability* (W. Mueller, Joffroy, and Solmon). This is a point, however, on which further observation is much needed. In a very chronic case of mine, which had arisen spontaneously, there was moderate emaciation and but slight diminution of electrical irritability. Everything probably depends upon the seat and the nature of the lesion.

Among the more rare, although not less interesting symptoms, we may mention the occurrence of *gangrenous bed-sores on the anæsthetized side* (Viguès, Charcot, Joffroy et Solmon), and the development of an inflammatory *knee-joint affection in the paralyzed leg* (Viguès, Joffroy et Solmon).

Finally, we must not fail to mention that the two last named observers, with the return of motility in their case, witnessed the occurrence of well-marked *ataxy.*

Pathological Physiology of the Disease.

Physiologists have instituted numberless experiments on the course of the motor, vaso-motor, and sensitive tracts, from the brain through the cord to the roots of the spinal nerves, and have especially and thoroughly ventilated the question of the crossing of the various tracts.

The course of the motor tracts was soon made clear, and there are no longer any serious differences of opinion with regard to this. There is no doubt that for the most part they decussate high up in the spinal cord and the medulla oblongata, in the pyramids, and the motor tracts for each side of the body run in that same side of the cord. Section of the right side of the cord, therefore, causes motor paralysis of the right extremities and the right half of the trunk.

The interesting results of Flechsig's researches[1] have shown even this relation to be only partly correct. It appears that the pyramidal tracts only in part pursue a crossed course (in the posterior lateral columns), and in part run their course uncrossed (at the inner portion of the anterior columns of the same side—[Hülsenvorderstrangband]). A vicarious relation exists between these two portions; sometimes the greater part of the tracts runs in the lateral columns and is crossed, sometimes in the anterior columns and is uncrossed. Consequently, very variable symptoms may arise on unilateral section of the spinal cord: complete paralysis on the side of section, or complete paralysis on the opposite side, or incomplete bilateral paralysis. The first of these conditions, according to pathological experience, is by far the most frequent.

What has been said of motor tracts is also true of *vaso-motor tracts*, at least to a great degree; indeed, more recent investigations have again proved that the principal mass of these tracts run their course in the lateral columns, and are uncrossed. A lesion of these tracts would therefore manifest itself upon the same side of the body. At the same time, their relations are somewhat complicated by the existence of the nerve-centres lying within the spinal cord itself, which have recently been demonstrated by Goltz and others.

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[1] *Paul Flechsig*, Die Leitungsbahnen im Gehirn und Rückenmark des Menschen. Leipzig, 1876. S. 291.

On the other hand, a difference of opinion long existed with regard to *the point of crossing of the sensitive tracts.* Brown-Séquard was probably the most positive in his assertion, based upon his numerous experiments, of their complete crossing within the spinal cord.[1] He stated that the sensitive tracts, conducting the sensations of touch, temperature, pain, and tickling, decussated with those of the other side very near their point of entrance into the spinal cord, and then ran to the brain in the opposite half of the cord. The only tracts which did not take part in this crossing, but ran through the cord on their own side of the body, like the motor tracts, were said to be those concerned in the phenomena of muscular sense (which enter the cord with the anterior roots). Brown-Séquard also gave other details which have thus far not proved of so much practical importance. According to him, the four different conducting tracts cross at different heights; those concerned in the sensation of temperature crossing somewhat earlier than the rest. During their farther course they lie separated from one another within certain definite segments of the cord, the sensitive tracts of the lower extremities being said to lie behind those of the upper extremities in the cervical portion of the cord.

These teachings were long combated by many. But in more recent times they have won universal recognition, especially on the basis of numerous pathological observations made on man. Even Schiff[2] has recently admitted their correctness for man, and it appears that part of the opposition to these teachings was due to the fact that the relations of these tracts is not the same in all classes of animals, but that incomplete decussations frequently occur. It has likewise been proved that centripetal fibres concerned in the production of reflex action also have a crossed course within the cord (Miescher, Nawrocki).

Section of one-half of the spinal cord would therefore cause anæsthesia of the opposite side for sensations of touch, pain, temperature, and tickling, while on the side of section these sensations would be retained, but that of muscular sensibility would be lost, which, in its turn again, would be retained on the opposite side.

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[1] Comptes rendus de la Soc. de Biolog. 1849. Gaz. hebdom. 1855. Nos. 31 and 36. Journ de la Physiol. de l'homme, etc. 1858. Vol. I. p. 176.

[2] Centralblatt f. d. medic. Wissensch. 1872. No. 49.

The accompanying diagram gives a representation of the course of the principal conducting tracts within the spinal cord. The motor and vaso-motor tracts (1 and 1′), which make their entrance, or rather their exit, through the anterior root (*v*), remain on the same side of the spinal cord. The same thing is true of the tracts concerned with muscular sensibility (2 and 2′). The sensitive tracts concerned in the sensations of touch, temperature, pain, and tickling (3 and 3′), which enter through the posterior roots (*h*), at once cross to the other side of the spinal cord, and pursue their course upward on that side. Section through the *right* half of the spinal cord at the height of *a* would then affect the motor, vaso-motor, and musculo-sensitive tracts of the right side (1 and 2) and the cutaneous sensitive tracts of the left side (3′).

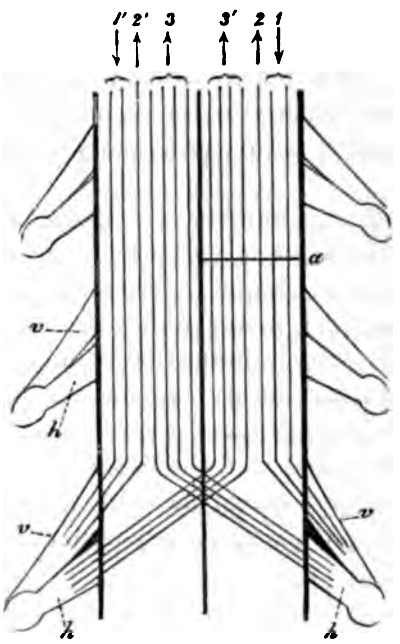

FIG. 13. Schematic representation of the course of the principal tracts within the spinal cord belonging to one pair of nerves. *v*, anterior, *h*, posterior root. 1, motor and vaso-motor tracts. 2, tracts for muscular sensibility. 3, tracts for cutaneous sensibility on the right side; 1′, 2′, 3′, the same on the left side. The arrows indicate the direction of physiological conduction.

Physiologists have furthermore discovered the fact that section of one-half of the spinal cord causes a high degree of *cutaneous hyperæsthesia of the same side*, extending to all varieties of sensation. This fact has not yet been satisfactorily explained. It is in part referred to the secondary inflammation of the cord following the wound, and the greater irritability of the sensitive tracts caused by this inflammation; and in part to the withdrawal of certain controlling influences which hold the irritability of the sensitive nerves within bounds, and whose tracts remain on the same side of the cord.

The *conditions of reflex action*, on unilateral section of the cord, do not seem to have received sufficient study at the hands of physiologists; we still lack definite teachings on this subject. The conditions seem to vary somewhat in different animals; at all events, the relations of reflex action to unilateral section of the spinal cord are not established with sufficient certainty to admit of our deducing a law therefrom. Nor do pathological observations on man lead to satisfactory conclusions on this subject. According to Woroschiloff's experiments it would appear that the fibres which control or check reflex action in a given lower extremity run their course principally in the same half of the cord, those which call forth reflex action principally in the opposite half of the cord.

At the same time we have a sufficient number of well-authenticated facts entirely to explain the most important manifestations of unilateral lesions. In case of simple unilateral section (for instance, at *a* in Fig. 13), we find motor and vaso-motor paralysis of the same side, together with a diminution of muscular sense, and a high degree of cutaneous hyperæsthesia. On the opposite side there is complete anæsthesia of the skin, while mobility and muscular sense are retained, and the fulness of the blood-vessels is normal. In fact, the correspondence of such cases with the results obtained in physiological experiments is quite complete.

If the unilateral lesion, however, is of somewhat greater longitudinal extent, if it extends over the domain of several entering roots, then another characteristic symptom arises in the form of an anæsthetic zone on the paralyzed side, lying above the hyperæsthetic region, and corresponding with the latter in width. The origin of this anæsthetic zone is easily explained. It is due to the implication of root-fibres of the same side *before* their crossing; the more of these implicated, the wider will be the anæsthetic zone; in progressive cases it may gradually extend over the entire trunk.

This relation may also be made clear by a simple diagram. Fig. 14 represents the course and the crossing of the sensitive nerve-tracts of five successive pairs of posterior nerve-roots. If we now suppose a lesion of the right half of the cord, extending over the shaded space, this will involve the sensitive tracts of the nerve-roots 1′, 2′, and 3′ of the left side as well as the sensitive tracts of all roots on the left side coming from still further back, thus causing a crossed, left-sided anæsthesia as high as the root 3′. It will, however, at the same time involve the sensitive tracts of the roots 2, 3, and 4 on the right side, and will cause anæsthesia within the domain of these nerves; this anæsthesia will therefore be zone-like, occurring on the side of the lesion, while all sensitive tracts entering behind the roots involved cross over to the left side unscathed, and continue their course undisturbed to the brain.

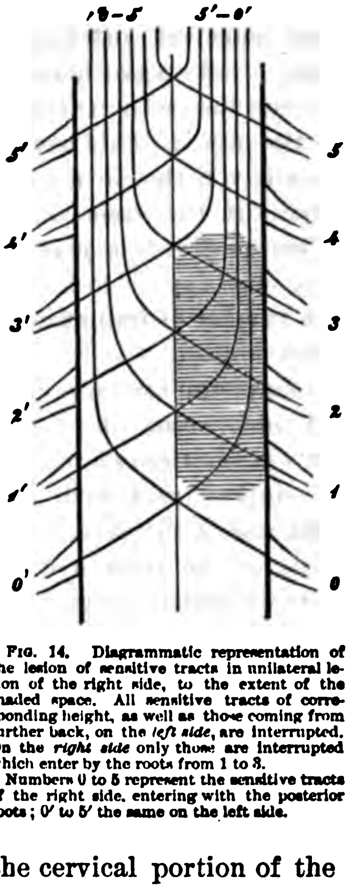

FIG. 14. Diagrammatic representation of the lesion of sensitive tracts in unilateral lesion of the right side, to the extent of the shaded space. All sensitive tracts of corresponding height, as well as those coming from further back, on the *left side*, are interrupted. On the *right side* only those are interrupted which enter by the roots from 1 to 3.

Numbers 0 to 5 represent the sensitive tracts of the right side, entering with the posterior roots; 0′ to 5′ the same on the left side.

The narrow hyperæsthetic zone in some cases observed above the anæsthetic belt—and, indeed, to a more marked degree on the side of lesion than on the opposite side—is explained by Brown-Séquard on the ground that the descending fibres of the posterior roots fall within the range of the lesion. It is a matter of no practical significance.

We have thus seen the characteristic manifestations of a unilateral lesion satisfactorily accounted for by known physiological facts. This is, likewise, none the less true with regard to certain manifestations which supervene when the seat of the lesion is very high, in the cervical portion of the cord. These manifestations consist, for example, of vaso-motor disturbances in the face, on the side of lesion, and the corresponding half of the head; in evidences of paralysis of the sympathetic in the neck, on the same side (contraction of the pupil, narrowing of the palpebral fissure, sinking of the eye in the orbit), etc.

Certain differences in the symptoms will occur according to the height, within the spinal cord, at which the lesion is situated.

We must not fail briefly to notice these, although, in fact, they follow, as a matter of course, from our knowledge of the physiology of the different divisions of the spinal cord.

In a *unilateral lesion of the lumbar enlargement of the cord*, in addition to the characteristic symptoms (paralysis and hyperæsthesia of the lower extremity of the same side, anæsthesia of that of the opposite side, etc.), an anæsthetic zone may be found on the paralyzed side, corresponding to the area of distribution of one or more lumbar nerves, being situated on the pelvis, the abdomen, the region of the groin, the anterior surface of the thigh, etc., thus constituting a zone which need not be exactly belt-like. By this means the anæsthesia may appear to be diffused over both lower extremities, but on careful examination it will be easy to recognize the true condition.

A *unilateral lesion of the dorsal portion* of the cord presents the most characteristic picture of symptoms, inasmuch as the sharp distinction between the two lower extremities, as regards their motor and sensory disturbances, stands forth with the greatest prominence. The hyperæsthesia and paralysis of the one, the anæsthesia and complete mobility of the other, appear in glaring contrast; the difference extends, in similar manner, to the trunk, ascending to a variable height; the belt-like anæsthesia of the paralyzed side becomes prominent, sometimes bounded by a narrow hyperæsthetic zone above (see Fig. 12).

In *unilateral lesions of the cervical portion* of the cord the relations are much more complicated, and often indistinct, especially if the lesion is just at the cervical enlargement. Aside from the manifestations in the face, the eyes, and certain cranial nerves, which, in this case, may complicate the picture of disease in an interesting manner, numerous varieties of the picture may be due to the higher or lower seat, and to the greater or less longitudinal extent of the lesion within the cervical enlargement. A little consideration shows how varied the motor paralysis in the upper extremity may be, according as the lesion involves the domain of the upper or lower roots of the brachial plexus, and how manifold the relations of hyperæsthesia to anæsthesia in the upper extremity of the same side may and must be according to the kind and the number of areas presided over by individual nerve-roots which are directly involved. The possible combinations that may here arise cannot all be cited, but if one has formed a clear conception of these relations, and mastered them in a simple case, he will also solve the problem in more complicated and difficult cases.

The symptoms presented in such a case are as follows: In the *lower extremities* and on the trunk the same conditions as in a lesion of the dorsal portion (paralysis, loss of muscular sense, elevated temperature, and hyperæsthesia on the side of the lesion; anæsthesia, normal temperature of the skin, no motor disturbances, normal muscular sense on the opposite side). In the *upper extremities*, *on the side of the lesion*, we find more or less muscles and nerve-domains paralyzed, hyperæsthesia in some or in all parts, mingled with anæsthesia of certain regions of skin or for certain varieties of sensation; on the *opposite side* no paralysis, more or less complete anæsthesia in all or in individual parts.

The *neck and head*, *on the side of the lesion*, sometimes show anæsthesia and hyperæsthesia of individual portions of the neck, manifestations of paralysis of the vaso-motor and oculo-pupillary fibres (higher temperature of that side of the head and body, heightened sensibility, narrowing of the palpebral fissure, contraction of the pupil, etc.); *on the opposite side*, sometimes anæsthesia of the neck (and a narrow zone of hyperæsthesia), a normal condition of the face and eye.

Naturally enough, this complicated group of symptoms is liable to manifold variations in individual instances.

As a matter of course, but little that is of general applicability can be said with regard to the *course*, *duration*, and *termination* of this malady, on account of the varying character of the anatomical changes which lie at the foundation thereof, and we must therefore refer the reader mainly to what has been stated in previous sections concerning wounds of the spinal cord, compression, chronic myelitis, sclerosis, etc.

The development of the characteristic group of symptoms may either occur quite suddenly, or may but slowly and gradually advance to a certain degree of completeness.

It then but rarely remains stationary and unchanged for any considerable length of time. The lesion generally extends further, in part through the transverse diameter of the cord, and in part longitudinally.

In most traumatic lesions the former change usually takes place early, the traumatic inflammation quickly spreading over the entire transverse diameter of the cord. Paraplegia at once follows, and we have before us the picture of transverse myelitis, which may end in death. Not rarely, however, the paraplegia again recedes, and we recur to the symptomatic picture of a unilateral lesion, which may then continue unchanged for many years. In not a few instances complete recovery has been seen to follow such traumatic unilateral lesions. It is singular that, according to Brown-Séquard, the disturbances of motion disappear earlier and more completely than those of sensation, the reverse of which holds good in case of peripheral injury of the nerves.

In the more chronic cases a similar course is maintained; the sclerosis or the tumor extends gradually over the entire transverse diameter of the cord; paraplegia occurs, followed by its resulting evils, and the further progress of the affection is determined by the fundamental process upon which it is based.

The lesion may, however, also extend longitudinally, gradually advancing upwards. The sclerosis or tumor may thus slowly creep upwards, and this does not usually occur without serious modification of the typical symptomatic picture, inasmuch as lateral extension usually goes hand in hand therewith. The final result is then likewise determined by the fundamental affection.

Diagnosis.

In view of the characteristic symptoms presented, the diagnosis of a unilateral spinal lesion is usually extremely easy.

The distinction between this and other forms of hemiplegia or hemiparaplegia is very simple.

Hemiparaplegia from *lesion of one s*
may be recognized by the fact that here
thesia are to be found on the same side,
erally affect only certain individual ner
extremities.

The *hemiplegic form of spasmodic s*
to be recognized by the absence of all di
therefore, also, of crossed anæsthesia, th
ance of the bladder, etc.

The unilateral spinal lesion may at
tinguished from *any form of cerebral*
that in the latter there is either no dist
it is to be found on the same side as the
one-sided paralysis of the face and of th
disturbances of various cranial nerves an
etc.

With regard to the *kind* of lesion t
times be difficult in non-traumatic cases
We can only attempt to overcome this
general pathological and diagnostic prin

Prognosis.

The prognosis of unilateral lesions is
form of disease lying at the foundation c
remarked of traumatic cases that they
favorable prognosis, as with proper ha
them end in recovery. Aside from thes
vidual cases, is the same with that of ot
the spinal cord, which are not localized c

Therapeutics.

Of course, there is no distinctive trea
spinal lesion. What concerns us is mer
of those spinal affections which are som
lateral half of the cord.

The principles and methods which ar

ment of wounds of the spinal cord, of compression of the cord, of chronic myelitis and sclerosis, of hemorrhages and new formations in the substance of the cord, of spinal syphilis, etc., may also be brought into play in corresponding cases of unilateral lesions.

15. *Acute Inflammation of the Gray Anterior Columns* [*Anterior Horns*].—*Poliomyelitis Anterior Acuta* (*Kussmaul*).—*Spinal Infantile Paralysis* (*von Heine*).—*Acute Spinal Paralysis of Adults.—Acute Atrophic Spinal Paralysis.—Paralysie Atrophique de l'Enfance* (*Duchenne*).

L. v. *Heine*, Beobb. über Lähmungszustände der unteren Extremitäten und deren Behandlung. 1840.—Ueber spinale Kinderlähmung. 2. Aufl. 1860.—*Badham*, Lond. Med. Gaz. 1836.—*Rilliet*, Gaz. méd. de Paris. 1851. p. 681.—*Rilliet* et *Barthez*, Traité clin. et prat. des malad. des enfants. Paris. 1853. Tom. II.—*Duchenne* (de Boulogne), de l'électrisation localisée. 1855. 2. éd. 1861. 3. éd. 1872.—Paralysie atroph. graisseuse de l'enfance. Gaz. hebdom. 1855.—*Vogt*, Ueb. d. essentielle Lähmung der Kinder. Bern. 1858.—*Bierbaum*, Die Paralyse der Kinder. Journ. f. Kinderkrankh. 1859. Heft 1 u. 2.—*Brünniche*, Ueb. die sog. essent. Lähmung bei kleinen Kindern. Journ. f. Kinderkrankh. 1861. Bd. 36. S. 366.—*Cornil*, Compt. rend. de la Soc. d. Biolog. 1863. p. 187.—*Duchenne* fils, Atroph. graiss. de l'enfance. Arch. génér. 1864.—*Laborde*, De l. paralys. dite essentielle de l'enfance. Thèse. Paris. 1864.—*Roger*, Paralysie infantile. Annal. de l'Eléctric. 1865.—*Prévost*, Compt. rend. d. la Soc. d. Biol. 1865. XVII. p. 215.—*Mor. Meyer*, Die Elektricit. in ihrer Anwendung u. s. w. 3. Aufl. 1868. S. 209.—*G. Salomon*, Zur Diagn. u. Therapie einiger Lähmungsformen im kindl. Alter. Jahrb. f. Kinderheilk. N. Folge I. S. 370.—*Bouchut*, Nature et trait. des paral. essent. de l'enf. Union méd. 1867.—*Johnson* and *Lockhart Clarke*, Extreme Muscul. Atrophy, etc. Med.-chir. Transact. LI. p. 249. 1868. —*Charcot* et *Joffroy*, Cas de paralys. infant. spin. avec lésions des corn. antér. etc. Arch. de Physiol. norm. et path. III. p. 134. 1870.—*Parrot* et *Joffroy*, Note sur un cas de paral. infant. Ibid. III. p. 309.—*Vulpian*, Cas d'atroph. musc. graiss. etc. Ibid. III. p. 316. 1870.—*Volkmann*, Ueb. Kinderlähmung und paralyt. Contracturen. Samml. klin. Vortr. Nr. 1. 1870.—*Duchenne* et *Joffroy*, De l'atroph. aiguë et chron. des cellules nerveuses, etc. Arch. d. Physiol. III. p. 499. 1870.—*Roger* et *Damaschino*, Rech. anat.-pathol. sur la paral. spin. de l'enf. Gaz. méd. de Par. 1871. Nr. 41 ff.—*W. Mueller*, Beitr. zur pathol. Anat. u. Phys. des menschl. R.-M. Beob. 2. 1871.—*A. Eulenburg*, Lehrb. der function. Nervenkrankh. 1871. S. 607.—*Dujardin-Beaumetz*, De la myëlite aiguë. 1872. p. 103.—*Hitzig* und *Juergensen*, Zur Therapie der Kinderlähmung. Deutsch. Arch. f. klin. Med. IX. S. 330. 1872.—*Petitfils*, Considérat. sur l'atroph. aiguë des cellul. motrices. Paris. 1873.—*Gombault*, Note sur un cas de paral. spin. de l'adulte suivi d'autopsie. Arch. de Phys. V. p. 80. 1873.—*Bernhardt*, Ueb. eine d. spinal. Kinderlähm. ähnl. Affect. Erwachsener. Arch. f. Psych. u. Nervenhrankh. IV. S. 370. 1873.—Beitr. zur Lehre v. d. acut. atroph. Spinallähm. Erwachs. Ibid. VII. S. 313. 1877.—*M. Roth*, Anat. Befund bei spin. Kinderlähmung. Virch. Arch. Bd. 58. S. 263. 1873.—*Bernheim*, Diction. encycl. des scienc. méd. 2. Sér. Tom. VIII. 1874.—*Mary Putnam Jacobi*, Americ. Journ. of Obstetr. June. 1874.—*C. Lange*, Hosp. Tid. 2. R. I. Aarg. 1874 (Virchow-Hirsch, Jahresber. pro. 1874. II. S. 164).—*Frey*, Ueb. temporäre Lähm. Erwachsener u. s. w. Berl. klin. Wochenschr. 1874. Nr. 1-3.—*Charcot*, Leçons sur les mal. du syst. nerv. II Sér. 3. fascic. 1874.—*Prévost* et *David*, Note sur un cas d'atroph. des muscles de l'émin. thenar, etc. Arch. de Phys. VI. p. 595. 1874.—*Seguin*, Spinal Paralysis of the Adult. Transact. New York Acad. of Medic. 1874. Nov.—*Erb*, Ueber acute Spinallähmung bei Erwachsenen u. s. w. Arch. f. Psych. u. Nervenkrankh. V. S. 758. 1875.—*Leyden*, Beitr. zur. pathol. Anat. d. atroph. Lähm. d. Kinder u. d. Erwachs. Ibid. VI. S. 271. 1875.—Klinik der Rückenmarkskrankh. II. S. 552. 1876.—*Weiss*, Ein Fall von acut. Spinallähm. bei Erwachs. Diss. Breslau. 1875.—*G. Burckhardt*, Physiol. Diagnost. d. Nervenkrankh. S. 174. 1875.—*F. T. Miles*, Case of Acute Spin. Paral. Transact. Americ. Neurolog. Assoc. for 1875. p. 217.—*M. Rosenthal*, Klin. d. Nervenkrankh. 2. Aufl. S. 409. 1875.—*Déjerine*, Note sur l'état de l. moëlle ép. etc. Arch. d. Physiol. VII. p. 253. 2875.—*Raymond*, Observ. de myél. centrale. Gaz. méd. de Paris. 1875. Nr. 18.—Deux cas de paralys. essent. de l'enfance. Ibid. Nr. 19.—*Wharton Sinkler*, Palsies of Children. Americ. Journ. of Med. Sc. April. 1875. p. 348.—*W. Sander*, Ueb. Rückwirkung d. spin. Kinderlähm. auf. die motor. Gebiete der Hirnrinde. Centralbl. f. d. med. Wiss. 1875. Nr. 15.—*Hammond*, Diseases of the Nerv. System. 6. edit. 1876. p. 451.—*F. Schultze*, Zur Lehre von d. spin. Kinderlähm. u. d. analog. Lähm. Erwachsener. Virch. Arch. Bd. 68. 1876.—*Eisenlohr*, Mittheilung üb. anatom. Befunde bei spinaler Kinderlähmung. Tagebl. der 49. Naturforschervers. in Hamburg. Beibl. S. 146. 1876.—*Couty*, Note sur un cas de myélite aiguë des cornes antérieures. Gaz. méd. de Paris. 1876. Nr. 18 u. 23.—*E. C. Seguin*, Myelitis of the Anterior Horns or Spinal Paralysis of the Adult and Child. New York. 1877.

Historical Sketch.

Jacob von Heine is doubtless entitled to be considered as the actual founder of the doctrine of acute spinal paralysis in children. In 1840 he wrote a monograph on the disease, and gave an exhaustive clinical representatien of the same, especially with reference to the atrophy and the deformities of the second period. It is true that some individual cases had been described before his time (by Underwood, 1784; Shaw, 1822; Badham, 1835; and others), but the knowledge of the disease had remained very limited, and its separation from other forms of paralysis during the age of childhood was not yet established.

This work of Heine's, marking an epoch, as it did, was followed by numerous discussions, and during the succeeding decades a succession of works was published on this disease (Rilliet and Barthez, Kennedy, Vogt, Bierbaum, and others), prominent among which are the manifold and extensive investigations of Duchenne (de Boulogne). They furnished an exhaustive clinical picture of the disease.

But the pathological anatomy of the affection remained obscure, and the strife as to whether this disease represented an "essential" or a spinal or peripheral paralysis was carried on more by theoretical arguments than by positive observations.

In the second edition of his work, in 1860, which is based upon a largely increased amount of material for observation, Heine declared very positively in favor of the *spinal seat* of the disease, which has ever since been universally designated as "*spinal* infantile paralysis." He thought it extremely probable that there was an alteration in the gray matter. Duchenne, though partly on other grounds than those of Heine, had likewise declared the spinal origin of the disease as in the highest degree probable. But at that time this view was not, as yet, supported by any positive evidence from the *cadaver*.

Indeed, such evidence remained lacking for some time yet, and, as we know now, could not be obtained until better methods for the microscopic examination of the spinal cord had secured general acceptance.

At the beginning of the previous decade workers in this disease, therefore, confined themselves almost entirely to clinical observations. The number of cases actually belonging to this disease was more accurately defined and limited, numerous details were more closely investigated, etc.

Cornil (1863) was the first to recognize distinct alterations in the spinal cord itself in this disease; but Prévost and Vulpian (1865) were the first to make the positive observation that the anatomical lesion was essentially situated in the gray anterior horns. This was confirmed by the observation of Lockhart Clarke in 1868. A very carefully investigated case of Charcot and Joffroy (1870) then stands at the beginning of a series of observations which, in all cases of spinal infantile paralysis, have, with perfect uniformity, demonstrated a disease of the spinal cord, and especially of the anterior gray horns. The existence of this lesion was established beyond all doubt by the cases of Parrot and Joffroy, Roger and Damaschino, Roth, Leyden, F. Schultze, Vulpian, W. Mueller, Déjerine, and others, and of late years no post-mortem has been made in this disease without the discovery of a similar lesion. It is true that there is not entire unity amongst observers as to the significance or the origin of the process, and that some apparently conflicting conditions have been discovered, but the fact is certainly established beyond all controversy that in this disease there is an acute lesion of the spinal cord, and principally of the anterior horns of gray matter.

It was not until about the time when the anatomical basis of the disease had been established that it was proved *not* to be exclusively an affection of early childhood, but one which might occur, in its typical form, during the later years of childhood or during adult life. This fact was first pointed out by Mor. Meyer, and was also confirmed by Duchenne, with a series of cases. Quite recently reports have accumulated establishing the occurrence of acute atrophic spinal paralysis in adults (Hallopeau, Gombault, Bernhardt, Frey, Charcot, Seguin, Erb, Weiss, F. Schultze, and others).

It is true that some cases have also crept into these reports with reference to which we may doubt whether they actually were *Poliomyelitis anterior acuta.* The line of distinction between this form of disease in adults and other forms of spinal disease, especially acute ascending paralysis, acute central myelitis, hæmatomyelitis, etc., can only be completed by further observations.

Definition.

This disease has very sharply defined clinical characteristics, as follows: It begins suddenly, usually with fever, with severe cerebral symptoms (deafness, coma, delirium, general convulsions); there is very rapidly developed and complete paralysis with entire relaxation of the muscles, this paralysis being of very variable distribution over the trunk and extremities, but generally in the form of paraplegia; there is an absence of any severe disturbances of sensation, no paralysis of the sphincters nor bed-sores.

A rapid improvement of the general condition soon follows; the paralysis proves not to be of a progressive character—indeed, gradual improvement of the same begins, although the restitution of movement is not uniform and remains in part lost forever. In some of the muscles there is extreme and rapidly progressing atrophy with degeneration of tissue; the development of the bones is retarded; the extremities are cold and cyanotic. During the further course of the affection considerable deformities of the limbs and trunk arise (club-foot, curvatures of the spine, paralytic contractures, etc.). The general condition of the individual is admirable, in spite of the permanent defects in the motor apparatus which almost invariably remain.

The disease may occur at all periods of life, though it is by far the most frequent in children between the ages of one and four years. It is susceptible of an unusually large number of grades of severity.

The *anatomical lesion*, although not yet quite certainly determined for all cases, may be regarded as most probably consisting in an *acute myelitis of the gray anterior columns* (anterior horns), which may extend more or less over the greater part of their entire length, but is disposed to be most heavily localized in the cervical and lumbar enlargements.

Etiology and Pathogeny.

There is undoubtedly a striking *predisposition* to this disease in the *age of childhood*, children between one and four years of age being by far the most frequently attacked by it. Duchenne, *fils*, even reports one case in a child twelve days old, and one in a child a month old, and the number of instances of the disease increases in the second half of the first year of life.

Various explanations have been given of this predisposition of the age of childhood. Probably the least satisfactory doctrine is to the effect that the physiological condition of the central nervous system in such young children of itself causes greater irritability and liability to disease of the same. C. Lange believes that the beginning of the exercise of voluntary movements at this age, especially those of walking, not unfrequently causes overexertion of the spinal cord, and thus induces a susceptibility to disease of the same. Dentition, however, has been more frequently invoked than anything else in explanation of this fact, and Heine in particular has emphasized the not rare occurrence of the symptoms of difficult dentition in his observations. At the same time, it is doubtful whether the disturbances which are so often associated with dentition do exactly determine the predisposition to disease of the cord. It is quite as reasonable to suppose that, the predisposition being already present, dentition merely serves as the exciting cause, and thus calls forth the disease. At all events, it seems that if dentition really stood in a causal relation to the spinal paralysis of children, the disease would necessarily be far more frequent than it is.

Another fact, which is now sufficiently established, viz., that of the occurrence of the disease *at later periods of life*, also goes to disprove the intimate connection of dentition therewith. In fact, the disease has been observed at all ages up to the sixties, although with much less frequency than during childhood.

Sex seems to exert no influence on the liability to the disease; it is about equally divided between the two sexes.

On the other hand, according to Wharton Sinkler, there seems to be no denying the influence of *seasons of the year*. Among fifty-seven cases collected by this observer not less than forty-seven occurred between May and September. Sinkler attributes this increased tendency to the disease during the summer months to the warm weather.

In the majority of instances there is no evidence whatever of any *hereditary influence* or neuropathic tendency. Duchenne even states that he never saw two cases in the same family. But Hammond reports its occurrence in two brothers, and Mor.

Meyer saw it arise simultaneously in twin brothers after measles. Duchenne, *fils*, reports a case in a child whose father afterwards died of tabes, but considers this as a mere coincidence. I have seen a similar case.

Neither do the *dyscrasias* often present in childhood (rachitis, scrofula, syphilis) appear to have any demonstrable influence in the production of this malady. Heine expresses himself very positively on this subject. On the contrary, it is just the most blooming, robust, and strong children that are most frequently attacked by the disease.

Our knowledge with regard to the *exciting causes* of acute spinal paralysis is just as scanty as that concerning the predisposing causes. In a large number of instances not the slightest cause can be assigned. Children are often attacked with the disease in the midst of the most blooming health, and physician and parents strive in vain to discover any cause therefor.

In a minority of instances the fact of *taking cold* has evidently been shown to be the cause. This has been repeatedly and undoubtedly demonstrated in children as well as adults. In Miles's case the disease appeared immediately after a cold bath, which the patient had taken while sweating, and after lively sexual excitement.

In other cases—and they are tolerably frequent—no other cause can be assigned than normal or difficult *dentition*. "The irritation of teething" has, from all time, been a favorite method of explanation for every conceivable trouble of infancy, especially those of a nervous character. At the same time, it is supposable that the circulatory and nervous disturbances which are undoubtedly often associated with dentition might develop this disease in a spinal cord predisposed thereto.

Traumatic influences of various kinds have repeatedly been regarded as causes, with what degree of justice it is hard positively to determine.

It is undoubtedly true, however, that the outbreak of the affection is not rarely connected with some *acute disease*. The entire symptomatic picture of acute spinal paralysis has repeatedly been seen to arise in children during the progress or soon after the subsidence of measles, scarlatina, varioloid, typhus, intermittent fever, etc. And the same thing has repeatedly been seen to occur in adults after the same diseases, or after severe puerperal disease. Authorities may conflict as to whether these attacks are to be ranked with those forms of poliomyelitis anterior acuta which arise spontaneously ; but there appears unquestionably to be the same localization of the disease, and in all probability a very similar anatomical process, in these well-defined "paralyses after acute diseases." We must trust to the future to settle this question.

Pathological Anatomy.

In spite of the numerous investigations of the past few years, the pathological anatomy of acute spinal paralysis can only be incompletely given. We are, no doubt, justifiable in laying down as a strict requirement that only those cases shall be considered as affording conclusive anatomical results *which have been accurately observed during life, and which have presented the well-marked clinical characteristics of the disease.* Many of the cases thus far described do not sufficiently fulfil this requirement. Many have been satisfied with the proof that there was paralysis during childhood without inquiring into the precise history of the beginning of the malady. Changes in muscles, bones, and nerves, which may notoriously be the results of very varied processes in the nervous system, have been accepted merely as the sequels of the "spinal paralysis of childhood." In this way probably very varied anatomical processes, not having the same significance, have been cited as belonging to this disease. When we see what diagnostic daring is developed in some authors by the desire to publish new observations on any rare form of disease, we shall be the more disposed to adhere to the postulate above uttered.

Secondly, we have a right to demand that only *the most recent cases obtainable* should be examined, with a view to determining the true essence of the pathological process present. Cases which are examined from twenty to sixty years after the fresh attack certainly cannot give safe and positive data for a judgment with regard to this disease. The literature of the subject shows that this requirement, too, has hitherto been but little complied with. The earliest anatomical examination which has thus far been reported (Roger and Damaschino) was made two months after the beginning of the disease.

I am convinced that it is only by the rigid observance of the two requirements above indicated, and by the careful criticism of observations, that we shall gradually succeed in learning the *essential* anatomical changes in this disease, and be enabled to separate them from those that are non-essential and accidental. It can hardly be otherwise than that a disease with so sharp and well-characterized a clinical picture should have some uniform anatomical lesion as its basis.

From the anatomical observations at present before us, it would at least appear most probable that this essential anatomical change consists in a *more or less diffuse acute myelitis of the anterior gray substance, which reaches its greatest intensity in the lumbar and cervical enlargements of the cord and, as a rule, leaves no permanent and irremediable alterations excepting at these two points.* In many instances, a moderate extension of the process, both in the gray substance and to the neighboring white (antero-lateral) columns, can be demonstrated, but probably this is only secondary and not essential.

The conditions which have been discovered, and upon which these views are based, must be divided into two groups, viz.: those which have been observed soon after the beginning of the illness (2 to 20 months), and those which have been observed very long after (17 to 61 years). Unfortunately, we are as yet entirely without any observations on the earliest stages of the disease (the first few days and weeks).

Observations made *at an early period of the disease* (Roger and Damaschino, Roth, Leyden's second case, Parrot and Joffroy, probably also the case very briefly reported by Rinecker[1] and examined by von Recklinghausen) show that sometimes nothing abnormal about the spinal cord can be discovered by the naked eye; sometimes the substance of the cord seemed tougher at the height of the cervical or lumbar enlargements, and there was a slight dwindling of the cord, especially at the expense of the antero-lateral columns. On making a transverse section, the markings were somewhat indistinct, the anterior gray substance was more or less discolored, whitish or reddish, sometimes soft and diffluent; sometimes a diminution can be shown in the volume of the gray anterior horns. The anterior roots—corresponding to the main seat of the disease—are gray, translucent, and atrophied. The membranes of the cord usually show nothing abnormal.

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[1] Jahrb. d. Kinderheilkunde. 1871. New series. V. p. 118.

Nothing short of a *microscopical examination*, however, reveals the exact seat, variety, and extent of the alterations. The principal trouble is found in the gray substance of the anterior horns, constituting *a tolerably diffuse lesion with regions of greater intensity* at the enlargements of the cord, particularly at the lumbar enlargement. Here we find, usually on both sides, though sometimes only on one side, an area of softening in the anterior gray horn of greater or less longitudinal extent (10 to 30 mm. or more, $\frac{4}{10}$ of an inch to 1$\frac{1}{8}$ inches), which is sometimes situated more towards the centre, sometimes more towards the front of the horn, and is separated from the surrounding parts by a more or less sharp line of demarcation. Similar areas are often also found in the cervical enlargement of the cord, more rarely at other points, in the dorsal or upper cervical portion. These areas of softening represent *inflammatory softening;* their substance is friable, soft, and disseminated with numerous granulation cells;[1] closer examination demonstrates an increase in the number of nuclei and in the amount of connective tissue and more than ordinary fulness of the blood-vessels. The most striking discovery, however, is the more or less *entire disappearance of the large, multipolar ganglion-cells;* those still remaining are found partly in all stages of degeneration and atrophy, partly, however, quite well preserved. It has not yet been possible to demonstrate any regular localization of these changes in definite groups of ganglion-cells within the anterior horns. The *nerve-*

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[1] In Leyden's case there were no granule-cells (Körnchenzeller); in the place of them there were numerous large, bladder-like, nucleated cells, like those of endothelium, in the gray substance, in part crowded together and in part more scattered, which, perhaps, had the same significance, in the morbid process, as the granule-cells.

fibres and *axis-cylinders* within the area of softening are also found to have *entirely disappeared.* In the immediate vicinity of the softening there is frequently quite a striking multiplication of nuclei, as though preparation were being made for a species of encapsulating process. The anterior horn, as a whole, appears diminished in size and wasted.

These areas, or foci, sometimes extend, by means of little prolongations, backwards, or towards the sides. The tissue surrounding them may be more or less completely or approximately normal, with well-preserved ganglion-cells. Generally, however, *slighter and more diffuse changes* can be demonstrated throughout a greater or less portion of the gray substance, usually throughout the entire dorsal portion, consisting of single granule-cells scattered through the tissue, the multiplication of nuclei, dilatation of blood-vessels, the disappearance of individual ganglion-cells. The accompanying diagram gives a clear picture of the localization and extent of the softening in the lumbar enlargement of the cord, according to Roth.

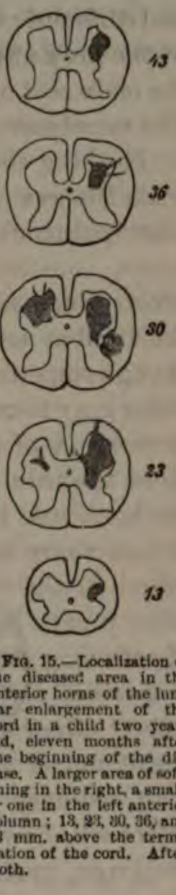

FIG. 15.—Localization of the diseased area in the anterior horns of the lumbar enlargement of the cord in a child two years old, eleven months after the beginning of the disease. A larger area of softening in the right, a smaller one in the left anterior column; 13, 23, 30, 36, and 43 mm. above the termination of the cord. After Roth.

At this stage there is generally not much to be seen in *the antero-lateral columns;* still they have repeatedly been proved to be diminished in size and the seat of a slight sclerosis. At other times a thickening of the trabeculæ has been found, atrophy of individual nerve-fibres (Joffroy, Damaschino), solitary granule-cells or Leyden's cells within the tissue; at all events, however, no very high degree of alteration takes place.

The *anterior roots* are diminished in size, atrophied, and under the microscope show the signs of degenerative atrophy.

In observations made *at a late period* the picture is, of course, radically different. We have such observations made between seventeen and sixty-one years after the origin of the disease by Cornil, Prévost, Vulpian, Lockhart Clarke, Charcot and Joffroy,

Petitfils and Pierret, Leyden, cases 1 an
F. Schultze, and others.

Now atrophy and shrivelling of certa
cord are much more evident, *even to the*
of the antero-lateral columns and the s
horns being especially prominent. Thes

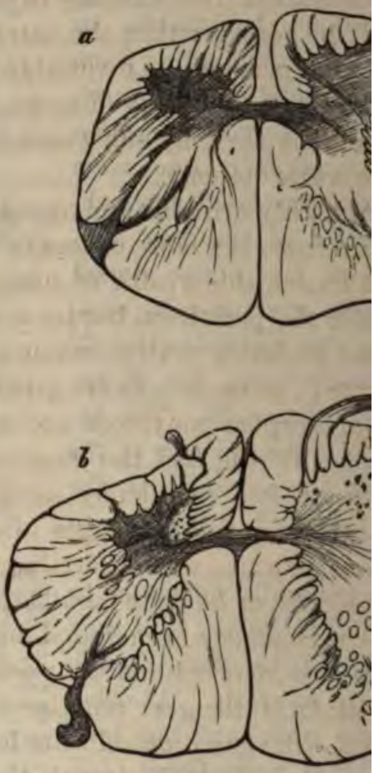

FIG. 16.—Spinal cord with Poliomyelitis anterior acuta, forty-th
case. *a*, Section through the lumbar enlargement; both anterior h
shrivelled, more on the left side than on the right. No ganglion-ce
largement; the left anterior horn and antero-lateral column very
The posterior columns and posterior horns in both sections are norm

in their dimensions in various directions,
so that the entire form of a transverse
well as its markings, seems to be chan
prominent if the lesion is limited to one
antero-lateral columns often appear som
cent; the posterior columns, on the con

The anterior roots are to a high degree atrophied, and the seat of gray degeneration.

On making a fresh transverse section of the cord there is frequently not much to be seen that is abnormal, aside from the alterations in form; after hardening, however, the principally degenerated areas generally appear with greater distinctness.

On *microscopic examination* the circumscribed foci first strike the eye; these will be found within the anterior horns at the lumbar and cervical enlargements, to a variable number and extent. In addition to this, in the majority of cases, there is also a more or less diffuse change in the gray substance, and a higher grade of alteration in the white columns.

The anterior horns are highly atrophic and shrunken; within the foci which they contain there is a more or less firm, fine-fibred connective tissue, rich in nuclei, this wealth of nuclei being not rarely most striking just in the peripheral border zone; thickening and enlargement, and probably multiplication of the blood-vessels, can be demonstrated; granule-cells are generally absent, but immense numbers of corpora amylacea are to be found, as well as pigment, etc. In the foci the ganglion-cells, and all nerve-fibres, are more or less completely destroyed. The ganglion-cells that may remain are in all stages of degenerative atrophy, pigmentary degeneration, and shrivelling. Well-preserved ganglion-cells are only to be found outside of the foci. "Clarke's columns" have generally been found intact; in two cases, however, they were also involved and their cells destroyed.

In the remaining divisions of the gray anterior horns—in the dorsal region, for instance—the ganglion-cells are less numerous than is normal, the connective tissue increased, there is a great wealth of nuclei, and numerous corpora amylacea.

In the antero-lateral columns a greater or less degree of sclerosis may be discovered, thickening and multiplication of the neuroglia, with or without pronounced atrophy of the nerve-fibres. This sclerosis may vary greatly in extent; sometimes it is confined to the immediate vicinity of the anterior horns of gray matter, sometimes it is distributed more diffusely over the entire antero-lateral columns, especially the posterior part of the lateral columns. It generally stands in most intimate relations to those

parts of the anterior horns which are mos
The anterior bundles of roots within the
as well as the anterior roots themselves,
picture of degenerative atrophy.

When we take a comprehensive view
we find, after all, that there is more uni
appeared on the first glance. It would
have our knowledge completed by some
days or weeks of the disease. But even
appears certain, that we have to deal es
inflammatory process in the anterior ho
do not know yet whether this is nothing
acute myelitis, or whether it is distinguis
peculiarities. This process is spread,
over the greater part of the gray anteri
greatest intensity in certain favorite spots
vical enlargements—and there causes dist
In these, besides the inflammatory chang
most striking and constant changes cons
struction and disappearance of the nerve
the multipolar ganglion-cells.

This process soon passes over into a
has entirely receded in some places, it
principal seats of the disease, to the dev
connective tissue, without any restitution
In the further progress of the disease, pro
as a rule, the antero-lateral columns ar
higher or lower grade of sclerosis. If th
veloped during the age of childhood these
retarded in their development; they ap
phied, and, together with the shrinking o
cicatricial contraction of the foci, cause
changes in form in the spinal cord. The p
terior columns almost always entirely esc

If all the anatomical conditions hitherto made p
spinal paralysis do not entirely agree with the repre
doubtless in part due to the circumstance mentioned
ous diseases have sometimes been brought into the

their admission being to prove that they were developed in childhood, and left behind them paralysis, muscular atrophy, paralytic contractures, and all sorts of deformities. But we know that various diseases of the spinal cord (multiple sclerosis, hæmatomyelitis, simple myelitis, etc.), provided they affect certain divisions of the gray substance, will produce exactly the same manifestations, and leave behind the same results. It would certainly be wrong to count all these as cases of acute spinal paralysis, for they are other forms of disease which happen to have a certain resemblance to it. For the present, therefore, we should be disposed to regard these unusual conditions as probably not belonging to this disease at all.

A question which has been raised by authors, and discussed with great liveliness, and which, it appears to us, cannot be determined with the material now on hand, is, whether the affection is originally a parenchymatous or an interstitial myelitis. Charcot, and after him Joffroy, Petitfils, and others have asserted that the primary step in the affection was the lesion of the large ganglion-cells, that, therefore, the disease is parenchymatous myelitis; while Roger and Damaschino, Roth, and others believe, on the ground of their examinations in fresher cases, that interstitial myelitis is the primary affection, and that the atrophy and disappearance of the ganglion-cells are secondarily caused thereby. In our opinion, no conclusive evidence has yet been obtained in favor of either view. We therefore decline to enter into any elaborate discussion of this difficult question, the more so as it is very elaborately treated of in the works of some of the authors above named, and as it does not yet appear to us to be of any great practical value, however much scientific interest it may possess. It is enough for our purpose to know that almost all authors agree on the fact of there being acute myelitis, and we may be content to leave it to the future to decide whether this myelitis is parenchymatous or interstitial; perhaps, as Dujardin-Beaumetz suggests, it may be decided that the connective tissue and nerve tissue are *simultaneously* attacked by the irritative process.

In those sections of the cord which are especially involved in the poliomyelitis, the *anterior roots* always appear very thin, gray, translucent, and atrophied. The microscope shows degenerative atrophy of the greater part of the nerve-fibres, well-marked proliferation and sometimes fatty infiltration of the interstitial connective tissue, thickening of the blood-vessels, etc.

The *peripheral nerves* have not yet been examined with the care that we could wish. The results of examination by electricity show with tolerable certainty that degenerative atrophy must exist at least in the motor filaments of many of these nerves. In a somewhat old case, F. Schultze found undoubted and considerable increase of the interstitial connective tissue with moderately numerous atrophic fibres. Déjerine also found atrophy of the nerve-fibres with increase of the connective tissue and nuclei.

The *muscles* of the paralyzed limbs always show the highest degree of alteration. Even with the naked eye the most varied grades and steps of such alteration can be recognized alongside of one another in one and the same patient.

In the first stages of the disease the muscles merely appear lean, atrophied, pale, and soft; later they become more grayish, or of a pale reddish yellow, being streaked partly with white connective tissue bands, and partly with intensely yellow bands of fatty tissue, which keep increasing more and more in number and in width. In the later stages the muscles acquire somewhat the appearance of decayed or withered leaves (welkes Laub), and finally they are entirely replaced by fatty tissue, which looks exactly like subcutaneous fatty tissue, and by its volume often more than restores the form and mass of the lost muscle. Another portion of the muscles dwindles into pure, connective-tissue fibrous strands containing no development of fatty tissue.

All these alterations take place to very varied degrees of extent; they may be seen irregularly intermingled in the various muscles of an extremity; not infrequently one may see several stages of change in one single muscle. The older the process is, the more will connective tissue and fatty tissue predominate, and in old cases some groups of muscles appear merely as great masses of fat.

The *microscopic examination* of these changes has unfortunately not been carried out in a rigidly systematic manner. But from what has been discovered by various observers, at the most different stages of the disease, thus much would appear to be certain, that the principal conditions found are those of *the various stages of degenerative atrophy of muscles*, as we have more particularly described it in another portion of this work,[1] that in addition to this, in some muscles, simple atrophic conditions and simple lipomatous conditions are found, but that these are of quite subordinate significance.

In the earlier stages of the disease, in the first months and years, we find at first a high degree of atrophy and wasting of the muscular fibres; in many of these the nuclei are greatly increased in number, the transverse striations are indistinct if not unrecognizable. At a later period there is cloudiness of the muscular fibres, at first caused by soluble molecules, afterwards by fat molecules; finally, the muscular fibres disappear altogether or almost so; individual fibres, however, or even whole bundles of them, may remain for a long time well preserved in the midst of the diseased muscle.

Even in the very first stages of the disease a simultaneous proliferation of the interstitial tissue begins; the width of the interstices is increased, they appear to be filled, here and there, with accumulations of nuclei, the walls of the vessels appear thickened and infiltrated with cells. This proliferation of connective tissue increases more and more; in all the later stages the muscle has scattered throughout it an abundance of firm-fibred connective tissue, rich in nuclei, so that some have spoken of it as a regular sclerosis of the muscle. When all the muscular fibres have finally disappeared from such a muscle, nothing but a firm, fibrous strand of connective tissue remains.

Sooner or later, however—and in youthful subjects this seems to occur comparatively soon—an abundant deposit of fat takes place in this interstitial tissue; whole rows of fat cells appear between the atrophied muscular fibres, which are in part found in a state of complete fatty degeneration; these rows multiply increasingly, overrun the entire tissue, and finally, after the destruction of the muscular fibres, the entire muscle is replaced by fatty tissue. This may go so far that the original volume of the degenerated muscle may be considerably exceeded by the volume of the fatty tissue that has taken its place. Thus a species of pseudo-hypertrophy of the muscle is brought about.

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[1] This Cyclopædia. Vol. XI.

To my mind it does not admit of the slightest doubt that the changes briefly described above represent the various and especially the latest stages of degenerative atrophy, as it is known and has been carefully studied especially in severe traumatic paralyses.

Even the *tendons* not rarely appear as narrow bands, atrophied, thin, and stretched.

The *bones* are always retarded in growth, both as to length and thickness, when the disease occurs during childhood; their epiphyses are stunted, the normal protuberances and processes less developed. Their external, hard lamella is thinned, friable, flexible, the medullary portion comparatively increased, its fatty contents more abundant (osteoporosis).

The *joints* are also strikingly lax, often regular dangling joints (Schlottergelenke), their ligaments thinned, stretched, and loose; the articular extremities are stunted, ground off, eroded; the articular cartilages are atrophied. The most manifold deformities are produced in this way, especially the severest forms and grades of club-foot. Extraordinary degrees of curvature of the spine are also not rarely produced in the same manner.

The *external skin* and the *internal organs of the body*, in the majority of instances, are quite normal, or only show such changes as are in no way connected with the disease of the spinal cord, and only belong to diseases accidentally acquired by the individual.

The *brain* is reported as entirely normal in almost all reports of post-mortems, and really seems to have nothing directly to do with this disease. Great scientific interest, however, is attached to an observation of Sander's, according to which the separation of the brain from a great part of the muscular apparatus of the body appears not to be without serious reaction upon certain motor divisions of the brain. In one case of "spinal paralysis of childhood" (which, it is true, was associated with "idiocy"), in a boy fifteen years of age, Sander found the two central convolutions and the *lobulus paracentralis* (which in man is supposed to be the seat of the so-called motor centres) materially stunted in their development. It is to be wished that there were more careful and more frequent examinations of this relation.

Pathology of Acute Spinal Paralysis.

Symptoms.

Hitherto this disease, as it occurs in children and as it occurs in adults, has almost always been treated of separately. This is surely not right; it is evidently precisely the same disease in both cases. To be sure there are certain variations in its picture of symptoms caused by the period of life, by the subsequent growth, by the different excitability of the organism of the child and the adult. For this reason a separate consideration of the symptoms at least would seem to be justified.

We will first delineate in detail the more important and frequent picture of the disease as it occurs in children, and add to this a short account of the same in adults.

a. Poliomyelitis Anterior Acuta in Children.

General picture of the disease.—The onset of this disease is marked by more or less *severe fever*, the significance of which may at first be entirely unrecognized. This sometimes follows certain premonitory manifestations (pain in the back and limbs, psychical changes, a tendency to be frightened, etc.), or may follow more or less difficult dentition, but most frequently it arises without any warning and while the child is in the midst of apparently perfect health. Very soon the heat and restlessness are followed by more *serious symptoms;* evidences of congestion of the head appear, giddiness, confusion of ideas, drowsiness, which may afterwards increase to somnolence and coma. Children that are old enough to tell of their sensations, complain of severe *pain in the back and in the limbs.* Not seldom general *convulsions*, of greater or less severity and extent, then arise, eclamptic seizures, which may occur once or repeatedly, but are generally of but short duration.

In many cases, however, such stormy manifestations are lacking, and the beginning of the disease is introduced merely by slight fever and a transitory ill feeling. Indeed, it is not rare for no initial symptoms to be noticed, and the characteristic paralysis may appear in the night, or during the day too, while the child is apparently perfectly well.

But even the most alarming symptoms generally disappear again in a very short time (from half a day or a day to three days), the fever subsides, the child's general health seems good, and one is inclined to think that the trouble is all past.

About this time the parents generally notice, with alarm, that a more or less extensive *paralysis* has imperceptibly crept on. This is made manifest when the child is taken out of bed, whe it is bathed, or when it is expected to walk. Either one limb o all the limbs hang down, relaxed and powerless, are incapable o motion.

It is but rarely that this is observed during the febrile an somnolent stage, and still more rarely that it has been possib accurately to follow the development of the paralysis. Th seems to progress pretty rapidly; one leg may be paralyzed the course of half an hour or of a few hours, the other leg f lows soon, then the arms; thus the paralysis may reach its f development during a night, or in from one to two days; it rare requires a longer time (a week).

Sometimes during the first few days there is also paralysis the bladder, usually incontinence, more rarely retention of urin this is of course hard to prove, with certainty, in little childr This may last a couple of days, or even weeks, but then alwa disappears.

So far as can be proved in children the sensibility of the pa lyzed parts is completely retained.

The further course of the affection is remarkably uniform different cases. The paralysis attains the maximum of its i tensity and extent very soon after the beginning of the diseas It does not increase any more, but merely decreases. The exte to which it may decrease varies very much in different instanc

In the rarest cases, improvement begins very soon, in from one to three weeks, being at first seen in single little movements, and then gradually extending further; the muscles at the same time remaining relaxed and soft, but not becoming much emaciated. This improvement advances further and further until complete recovery, which may be attained in the course of a few months. These are the cases of so-called temporary spinal paralysis.

Generally, however, the course is not so favorable. It is true that improvement usually begins early, but it only advances to a certain point; the arms may become entirely free, or perhaps only partly so, the movements of the shoulders and elbows may return while the hands remain partly paralyzed, or *vice versa.* In the same way partial recovery occurs in the lower extremities; one limb may become entirely movable while the other remains completely paralyzed, or the thighs may return to their normal condition while the legs remain paralyzed; indeed, single groups of muscles or even single muscles may again acquire the power of motion or remain completely paralyzed, in the most multiform manner.

In the great majority of cases the larger part of the muscles thus remains paralyzed for a very long time, or permanently. And in all these muscles a *high grade of rapidly progressive atrophy* is at once set up, and examination by means of electricity gives well-marked evidences of *degenerative change.*

The limbs so severely paralyzed now present a very characteristic condition. There is complete paralysis and entire flaccidity of the muscles, a high degree of atrophy of the same with great softness to the touch, total failure of all reflex action in the limbs (both of the skin and tendons); the sensibility of the skin is everywhere normal, the patient does not take to his bed, the skin is strikingly cool, bluish, cyanotic, and not seldom somewhat bloated.

Children then remain for a long time in this condition, which may extend more or less widely over the trunk and extremities, and may be well developed within a few weeks after the beginning of the illness. Their general condition is excellent, the vegetative functions are entirely in order, the brain is perfectly ally take place, changes which are peculiarly c the affection, and even in later life betray, to th at the first glance, the disease which was gone t childhood. These changes are as follows:

Progressive emaciation of the muscles, up to complete disappearance of the same, and a skel tion of the limbs; not rarely, however, there i production of fat in the same, whereby the atr great degree, be hidden.

The development of paralytic contractions in muscles and groups of muscles.

Retardation of the growth of the bones; short ing of the limbs; distortion, laxness, and unusua joints, displacement of articular extremities of b

As the result of all these changes the mos extreme deformities of the limbs, the joints, the etc., are produced. Club-foot, which is of such rence, loose dangling joints, and extreme degrees ture, usually owe their origin to infantile spinal shrunken, paralyzed, crippled members, hideou capable of any use, constituting a burdensome a body rather than a necessary portion thereof, ap contrast to the well-developed, well-nourished m

In later life these patients drag their defor weaknesses around with them, without any re ever taking place. Not infrequently, however, provement occurs because the patients grad

This of course varies very much, in individual instances, according to the extent and seat of the lesion; sometimes but one lower extremity is paralyzed and shortened, sometimes both; or the arms alone may be affected, sometimes only one arm, or sometimes even parts of one extremity alone. The most wonderful curvatures and deformities thus occur (they are very graphically depicted in the plates of Heine's book); the most striking cripples on the public streets, the chronic inmates of orthopædic hospitals, the most grateful objects for the ingenuity and art of those interested in mechanical surgical appliances, are, in great part, the victims of the spinal paralysis of childhood.

The duration of life does not seem to be in any way influenced by the disease; its victims, aside from their deformity and paralysis, remain well and fresh, and may reach old age. They fulfil their calling, so far as they are capable of having a calling, get married, have children, and comport themselves like healthy men and women, with the exception of the defects left behind by the disease of their childhood.

There does not even seem to be any increased disposition remaining to further disease of the spinal cord. I have thus far met with but two cases in which adults, who showed signs of the acute poliomyelitis anterior of which they had suffered in childhood, were attacked with spinal disease (in one case with tabes, in the other with sclerosis lateralis amyotrophica). Raymond saw progressive muscular atrophy developed at a later period in such an individual. In one case I afterwards observed the development of epilepsy.

Value of the Individual Symptoms.

The *initial fever* has been but very little examined and investigated as yet. It may be very insignificant or very severe. In some, although rare instances, it appears to be entirely lacking; according to Duchenne, Jr., this is claimed to have been true in seven cases out of seventy; the correctness of this statement does not, however, seem to have been tested by the thermometer. It is certainly present in the majority of instances, although of very varying intensity and duration. It usually lasts but one or two days, sometimes only half a day or a few hours; rarely for from six to eight days, or even fourteen. It is not yet determined whether the fever stands in any definite relation to the intensity and extent of the disease in the initial stage; but it certainly appears as though the intensity of the fever bore no definite relation to the extent of the disturbances that are left behind.

Probably no one would dissent from the view that the initial fever is simply of an inflammatory nature, and is dependent on the acute process localized in a large part of the spinal cord.

In most instances the *initial brain symptoms* are very prominent. They also appear in extremely varied degrees of intensity; sometimes the outbreak of the severer manifestations of disease are preceded for a few days by light nervous symptoms; greater psychical irritability, unusual liability to fright, uneasy sleep, gritting the teeth during sleep, frequent rolling of the eyes, paroxysms of terror, and the like, are noticed in children. With the beginning of fever, confusion of ideas with slight somnolency is generally soon observed; all sorts of delirium, sometimes light and sometimes active, appear; occasionally there is profound unconsciousness.

But the most striking and alarming manifestations are the convulsions which occur in many cases, and which may be of a light or a severe form. If light they may be confined to convulsive twitchings of the face and extremities, to grinding the teeth and rolling up the eyes; if severe they consist in an outbreak of general convulsions, which remind one of eclampsia, but which are generally repeated but a few times, and not rarely terminate with the first attack. This symptom also is liable to great variation; sometimes the attacks only last one or two hours, at others they occur frequently during one or two days; the individual attacks are sometimes lighter, sometimes more severe; they may be accompanied with vomiting. But although they often look very threatening, they do not appear to cause any immediate danger to life. It remains to be proved, however, whether the convulsions, which carry off some little children within a few days, may not sometimes belong to the initial stage of acute spinal paralysis.

Finally, however, there are also numerous instances in which severe initial brain-symptoms can be demonstrated, in which, at all events, all convulsive manifestations are entirely lacking.

The pathogenetic explanation of these initial brain-symptoms has not yet been found. It may appear doubtful whether they are to be regarded as mere manifestations of fever; it is not proved whether the sudden irritation of a large part of the spinal cord makes an impression on the brain of a child sufficiently powerful to induce such severe disturbances. Neither has there as yet been furnished any demonstration of a simultaneous lesion of the brain, during the initial stage; this would have to be of a very transitory nature; and it certainly is not plausible to suppose, like Frey, that this lesion consists in a cerebral hyperæmia which is induced by paralysis of the spinal vaso-motor tracts for the cerebral vessels, and which disappears again with the speedy abatement of the inflammatory swelling of the cord. Perhaps a number of these causes work together to cause the remarkable occurrence of severe initial brain-symptoms in this spinal disease.

Unquestionably, however, the essential symptom of the disease is the *paralysis with subsequent atrophy of the muscles.* This lends to the picture of the disease its main peculiarity, and is almost exclusively responsible for the severe sequels of the affection.

The *development* of the paralysis often occurs quite unnoticed. It is not until the febrile manifestations have subsided, when the child wants to use its limbs, when it is to walk or stand, when it is bathed or dressed, that one notices the paralysis which has in the meantime crept on, and even then it is not unfrequently looked upon as being nothing more than the lassitude resulting from the fever. This error is naturally cleared up by the further course of the affection. Children of a somewhat older age often notice the paralysis earlier and call attention to it in good time.

The paralysis is almost always developed with great rapidity, and appears well-nigh simultaneously in all the parts attacked. It does not, however, come on with the quickness of apoplectic paralysis, but several hours always pass, half a day or a night, before it reaches its maximum. In some few cases a slower spread of the affection is observed, so that it may not reach its acme for several days. Again, it has been seen to arise in several successive onsets, so that the several extremities were attacked at intervals of eight days; each single onset, however, then goes on with a certain degree of rapidity.

Whatever the precise method of development may be, this much is certain, and is to a high degree characteristic of this very disease, *that the paralysis reaches the maximum of its extent and intensity at the very beginning of the disease, or at least within a short time thereafter;* that from this time on it advances no further, but merely recedes. The paralysis, therefore, has distinctly no progressive character.

The paralyzed muscles now appear entirely flaccid and soft, and show no trace of contraction; the limbs are capable of free passive motions in all directions; they hang on the body like limp, dead masses. The turgescence of the muscles appears at the same time to be diminished, likewise the turgescence of the skin. Reflex action is completely extinguished in the paralyzed muscles; no reflex action can be aroused by the strongest irritation to the skin or tendons, although this irritation may be very distinctly felt by the little patients.

The *distribution of the paralysis* is extraordinarily different in different cases. The lower extremities are almost always attacked; sometimes, however, the upper extremities are seized at the same time, as well as a great part of the muscles of the trunk, especially those of the spinal column, and sometimes also of the neck. Not rarely but *one* lower extremity is attacked, and sometimes the paralysis occurs in the form of hemiplegia, the arm and leg of the same side being affected. This form, the existence of which was denied by Heine, has been observed by Duchenne, is declared by Volkmann not to be at all rare, and was found by Sinkler eight times in eighty-seven cases. Duchenne saw one case of crossed paralysis in the arm and leg. It is rare for but one arm to be totally or partially paralyzed, still more rarely does the paralysis seem to be confined to the upper extremities without participation of the lower. It is very common, however, to meet with a quite partial paralysis of single muscles and groups of muscles, especially on the lower extremities, and, at all events, in the majority of cases, even where the paralysis is exceedingly wide-spread, there is a very non-uniform implication of the various groups of muscles and even of single muscles in a given nerve-domain. Hence arises such a manifold variety in individual cases as to defy all description, and we must therefore refer our readers for details to the reports of cases accumulated in literature. We merely mention, by way of example, that in the domain of the peroneal nerve the tibialis anticus not rarely occupies an exceptional position, either being paralyzed alone or alone free; that in the domain of the crural nerve the sartorius or the tensor vaginæ femoris often give the same history; that in the domain of the radial nerve the same is true of the supinator longus; that the deltoid is not rarely attacked by paralysis alone, and other instances of the same kind.

Very soon, sometimes within a few days, but generally not until within the next few weeks, a gradual *improvement of the paralysis* sets in, affecting a greater or smaller part of the muscles involved, while the rest usually remain paralyzed permanently or, at least, for a long time. This very symptom of the partial restitution of muscles is also very characteristic of the disease. Very rarely this restitution is complete, extending to all the paralyzed muscles, so that the improvement once begun advances to complete recovery (so-called temporary spinal paralysis).

The upper part of the body, and the upper extremities generally, improve soon, and are the parts most likely again to regain their full activity; a part of the muscles of the trunk also almost always takes part in the improvement, while others, particularly the muscles of the back, remain permanently paralyzed. In the same way a partial restitution often takes places in the lower extremities; one limb may become entirely or partially capable of performing its functions again, while the other remains paralyzed; it is very common for single portions of an extremity to remain permanently paralyzed, often single muscles alone; this, too, afterwards gives great variety to the picture of the disease.

As a general rule, the progress of this improvement only lasts for a time; this progress is most manifest during the first four to eight weeks, and then often inspires delusive hopes. At a later period the progress is slower, and after six or nine months from the beginning of the disease there is no question of any further spontaneous improvement; if any is then observed, it is as a rule only apparent, caused by use and practice of the restored or unaffected muscles. Doubtless, however, during the later stages of the disease, too, appropriate (electrical) treatment may still effect improvement in some muscles, which is sometimes very striking, although generally but slight.

In almost all muscles whose mobility is not very soon restored, and which remain more or less permanently paralyzed, a well-marked and *rapidly progressive atrophy* very soon appears. This begins in the first weeks of the disease, and even those muscles that are but slightly affected generally give unmistakable evidence of a slight degree of emaciation; this soon disappears again, however, on the restoration of mobility. In the muscles more severely affected the atrophy is generally very distinct in the course of a few weeks. The muscles become more and more flaccid and attenuated, and may so completely disappear that the skin seems to lie immediately upon the bone, and the limbs assume a skeleton-like appearance. During the further course of the affection the volume of many of these muscles is again increased, so that there appears to be a muscular mass present again; this is caused, however, by the secondary deposition of fat in the connective tissue, and it is generally easy to prove, by the test of galvanism, that not so much as a trace of muscular tissue remains in these pseudo-muscles. In some cases the atrophy of the muscles is completely masked from the very beginning through the more abundant development of fat in the subcutaneous connective tissue, or by early deposits of fat in the muscles themselves; this is especially true in well-nourished, well-developed children. In such cases, however, advanced atrophy is always to be recognized by the extreme softness and flaccidity of the muscle as compared with one that is healthy, even if the tape-measure indicates no material difference in comparison with the healthy side.

Another sign which may be mentioned as being often observed in adults, although more rarely established in children, is that during this rapidly advancing process of atrophy the muscles are quite sensitive on pressure.

The alterations in the electrical irritability of the motor nerves and of the muscles, which are always to be found in the spinal paralysis of childhood, stand in most intimate relations to atrophy of the muscles. The earlier authors, it is true, only tested the relations of faradic irritability, and Duchenne, in particular, is entitled to the credit of having carefully investigated this in every direction, and of having established important diagnostic and prognostic rules. Duchenne found that in muscles which are severely affected, *faradic irritability begins to sink very quickly*; that it is materially diminished at the end of three to five days, and entirely extinguished by the seventh day or during the course of the second week. All the paralyzed muscles, however, in which this does not occur during the course of the second week, in which faradic irritability is only more or less diminished, but not completely lost, do not remain permanently paralyzed, but regain their mobility, and do so the more promptly the less their faradic irritability has been diminished. These statements have been confirmed by all subsequent observers.

But our knowledge on this subject did not widen nor gain in precision until the paralyzed nerves and muscles were also tested by the galvanic current. Then it became at once manifest that the loss of faradic irritability found by Duchenne was nothing else than a part of the phenomena of *degeneration*. Indeed, as might have been expected, *a priori*, in view of the degenerative changes in the nerves and muscles, the alteration in electrical irritability which seems to be always capable of demonstration, in its various stages in the spinal paralysis of children, constitutes *the reaction of degeneration*. So far as I know, Salomon was the first to establish the characteristic alterations of galvanic irritability in the muscles in the spinal paralysis of children, and since that this alteration has often enough been observed again, though it has also often enough been overlooked. The course of the alteration is about the same as that in severe traumatic paralysis. There is rapid loss of faradic irritability in nerves and muscles, either permanently or, at least, for a long time, that is, until

mobility returns; and then the familiar
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by a fortunate chance, and the examination of th
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are too rare to furnish sufficient material for observati
in large children's hospitals. I regard the *reaction of*
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tinctly shown only in the first years of the disease,
with certainty at a later period. I have, at least, ne
ber of cases, failed to find the reaction of degener
above statement is based upon the examination of
eight, and fourteen weeks, after six, twelve, twenty-

and from that up to several, or even many years. Furthermore, it also entirely corresponds with the known results of electrical examination in the same affection in adults.

The *changes in the bones and joints* which arise in the course of the disease are almost equally important with the changes just described in the muscles. The bones of the paralyzed limbs are materially retarded in their longitudinal growth, so that, in the legs for instance, differences of from 3 to 20 cm. (1.1 to 7.8 inches) may occur, as compared with the healthy side. The same thing is true in the upper extremities. The long bones, furthermore, remain much thinner than normal; they not rarely become porous and inclined to bend, yielding, and friable. Their epiphyses and processes grow smaller and less distinct; the shoulder-blade and the patella often remain far below their natural size, the paralyzed hand or foot is shorter, narrower, and thinner than the sound one; even the pelvis may remain considerably behind-hand in its development, being below the normal measurements of circumference and height.

It is a remarkable fact that the interference with the growth of the bones is by no means always in direct proportion to the atrophy of the muscles of the same extremity; sometimes the one and sometimes the other is in the ascendancy, and the interference with the growth of bone may persist in spite of the disappearance of the paralysis (Volkmann). I have myself had occasion to observe this fact.

Volkmann also, not without reason, counts a case of "unilateral paralysis of the face," observed by him, as belonging to the spinal paralysis of childhood. Other cases should be tested with reference to similar connection.

The *joints* become deformed, abnormally movable, partly through disappearance of the articular extremities of bone and of cartilages, partly through relaxation and stretching of their ligamentous apparatus. The movableness of the limbs is thus often rendered very great, so that patients can frequently execute all kinds of unusual movements and wonderful dislocations of the limbs.

The *changes in the skin* must also be noticed. The skin becomes flaccid, withered, doughy, sometimes dry, scaly, brittle The most striking characteristic, however, is the *coldness an cyanosis* which is uniformly observed in the paralyzed limb Heine carefully measured the lowering of temperature in man cases, and was able to prove very marked differences as con pared with the healthy extremity. There can hardly be a dou that this lowering of temperature and cyanosis are due less diminished nutrition in the paralyzed limbs than to vaso-mot paralysis, and it would be interesting to note whether, in the fi stages of the disease, there is not a temporary elevation of te perature. I demonstrated this to be the case in one adult p tient.

By means of the conditions above described—the paralys and atrophy of muscles, the interference with the growth bones, the changes in joints—all severer cases lead to *paralyt contractures* and *secondary deformities*, which may cause t most revolting distortions of the limbs, of the spinal colum etc., and are most graphically set forth by Heine in numero illustrations. The atrophy and secondary contraction of t muscles, the laxness and malformation of the joints, the curv tures of the spine, all combine to produce the most deplorab and extraordinary cripples, such as, in countless numbers, exci the sympathy of the passers-by upon the highways and in t market-places.

What are called *paralytic contractures* are especially effec ive in the development of these deformities. According t Heine, these do not begin to develop until the children com mence to use their limbs and drag themselves about with them whether well or ill. This statement, however, may not be s rigidly correct.

The most varied forms of club-foot occur (talipes varus, va gus, equinus, and calcaneus), and various combinations of th same (most frequently of the equinus and varus). Retrocurv ture of the knee, inversion and eversion of the same, are also me with, as well as contractures in the knee- and hip-joints, kyph sis, lordosis, and extreme instances of scoliosis. In the uppe extremities the deformities are liable to be much less serious the hands and fingers are generally found flexed, the shoulder joint gradually becomes immovable, the pectoralis and the latissimus dorsi are more or less contracted.

The occurrence of contractures is chiefly the result of a high degree of paralysis and degenerative atrophy of the muscles. Considered in detail, the more precise method of development may be threefold.

1. By far the most frequent cause of paralytic contracture, in these cases, is the *continued approximation of the points of attachment of the muscles*, induced partly by the weight of the parts, partly by external pressure in walking, standing, sliding, etc. This point has been thoroughly discussed by Volkmann, on the basis of Hueter's investigations, and the former seeks to explain almost all paralytic contractures on mechanical grounds. According to him the deformities are developed partly by reason of the weight of the limbs concerned and the position which they assume when at rest, partly through the fact that limbs, articular extremities, and articular surfaces are subjected to an abnormal burden, when called into use, and are thus gradually pressed into abnormal positions. In both instances the points of attachment of certain muscles would be more or less permanently approximated to each other, and thus a gradually increasing nutritive shortening of the same occurs, a permanent contracture and deformity.

This must certainly be the explanation of all those contractures and deformities which arise when all the muscles surrounding a joint are completely paralyzed, or when the deformity and contracture take place in the line of traction of those very muscles which are especially paralyzed. Talipes equino-varus is the form most easily explained; the foot, being left to its own weight, assumes this position, which becomes more and more fixed the longer the patient remains in bed, or the longer stepping upon the foot, which would counteract this deformity, is avoided, or the patient merely steps upon his toes.

Paralytic talipes valgus occurs when the patient begins to walk again very soon after his attack, and steps on the foot while there is more or less complete paralysis of the muscles of the leg; he then steps on his entire sole, and the foot, which is loose in its joints, gives way, toward the outer side (knickt soweit nach aussen um), until prevented, by physiological restraints, from bending any further. Even talipes ca caneus is said to be due principally to mechanical cause inasmuch as when the muscles of the calf are entirely par lyzed, the individual steps upon the os calcis, and this is the gradually and mechanically pushed forward ; by this mea the distance between the points of attachment of the muscles the sole is also diminished, and for this reason the plantar curv ture of the sole reaches a peculiarly high degree of developme in this variety of club-foot. It appears to us, however, that he the co-operation of the antagonistic muscles of the front of th leg, which are less paralyzed, is of material significance. Retr curvature of the knee is brought about by the fact that the join deprived of its power of fixation by the muscles, is extende backwards as far as its physiological restraints will admit, i order to transform the leg into a firm support for the body. I the upper extremities, too, the majority of contractures and d formities are developed in the same simply mechanical manner.

2. But contractures may also furthermore be brought abo by the fact *that the antagonists of the paralyzed muscles rema effective.* In every voluntary action of the same their points attachment are approximated to each other, and there is no mu cular power present to draw them apart again ; in many case indeed, this is accomplished through the weight of the parts other mechanical means ; but where this is not the case the muscles fall into permanent nutritive shortening, and thus i crease the deformity. A certain degree of participation in t production of paralytic contractures and deformities, even thoug it be but subordinate, must certainly be attributed to this acti mobility of antagonistic muscles ; thus in talipes calcaneus, th in contractile bending of the knee-joint with complete paralys of the quadriceps extensor muscle.

3. Finally, a certain part in the production and fixation paralytic contractures may be attributed to *the proliferation interstitial connective tissue and its subsequent retraction,* whic takes place with the degenerative atrophy of the muscles. Thi however, would seem to be of subordinate significance.

Mechanical causes certainly play the principal rôle, in thi matter, and the manifold and adventurous methods of locomotion to which many of these patients accustom themselves, as well as the extremely varied localization and extent of the paralysis, account for the fact that the most wonderful and striking malformations and crippled conditions occur in these very patients.

So far as concerns the *pathogenetic explanation* of the principal symptoms of the disease, as described thus far, these may doubtless all be explained in one way. The acute, inflammatory, and destructive lesion of the gray anterior horns is undoubtedly to be held accountable for all these disturbances. And indeed the lesion of the large ganglion-cells—and, we may add, the destruction of all nervous channels of communication—seems to be of very special importance in this respect. It is difficult to make any more definite statement on this subject; still the application of what we now know about it seems pretty easily made; inasmuch as the spinal paralysis of children is one of the very conditions, among others, which has contributed to clear up our knowledge of the physiological functions of the gray anterior horns.

According to all we know, the gray anterior horns are points of passage for direct motor conduction from the lateral columns to the anterior roots. Whether this conduction, in all cases, passes through large ganglion-cells, and whether these, therefore, constitute a species of ganglionic centres (Knotenpunkte), is not yet certainly determined, but seems very probable. It seems, furthermore, tolerably certain that the large ganglion-cells exercise distinct trophic functions with reference to the nerves and muscles (and probably also to the bones, joints, etc.). It is still undecided whether all these ganglion-cells have at the same time motor and trophic functions, or whether, as Duchenne and Joffroy conjecture, and as Hammond also believes, there are two kinds, purely motor and purely trophic; nor is this point capable of easy demonstration. At the same time the latter supposition is not exactly improbable, and is certainly more convenient as applied to the theory of this and kindred diseases.

The more or less complete destruction of the anterior gray horns, arising acutely, explains all the manifestations encountered in quite a plausible manner: the paralysis by the destruction of motor tracts, the atrophy of muscles and the hindrance of the growth of bones, with their results, by the destruction of the trophic apparatus. The fact of the disease occurring in certain circumscribed areas, and the immunity of certain groups of ganglia, explains the localization and extent of the paralysis, and the immunity of certain single muscles and groups of muscles. The acute inflammatory nature of the process accounts for the sudden appearance of the paralysis; the wide extension of the process over the spinal cord accounts for the initial fever and the wide extent of the paralysis at the beginning. The possibility of the partial resolution of the acute inflammation explains the rapid disappearance of the first stormy manifestations, and the later partial restitution of the muscles.

Upon the supposition, therefore, that our views concernin the functions of the anterior gray horns are correct, the clinica manifestations of the spinal paralysis of childhood are in sufficiently satisfactory correspondence with the anatomical lesion.

The remaining functions of the spinal cord, with few exceptions, suffer no disturbances whatever, or but very slight ones.

The *sensibility of the skin* is almost always intact. At th beginning of the affection there may be complaints of pain, pa æsthesia, and the like, here or there, but they are quite trans tory manifestations which may easily be explained by the inflammatory swelling and hyperæmia in the initial stage. At a lat period sensibility is generally intact everywhere. Only occasio ally is there a slight, generally very insignificant dulness of se sibility complained of, especially a slight diminution of sensibi ity to pain and lessened intensity of the sensation of ticklin This may be explained partly by an unusual extension of th diseased process to the central and posterior gray substanc partly, however, also by the great coldness and poor nutritio of the extremities.

Reflex activity, on the other hand, always suffers very sever disturbances. It is a matter of course—on account of the dege erative atrophy of the muscles and nerves, if for no other rea son—that in all muscles which are severely attacked reflex actio is completely extinguished; in these muscles no sign of refle twitching can be aroused, either through the skin or throug the tendons. But even in those muscles which are but slightl attacked, reflex activity is liable to be, at least temporarily, very much lowered or entirely extinguished. As the reflex circuits are generally considered as being located in the anterior gray substance, and reflex activity is thought to be connected with the large ganglion-cells, this condition of things is readily to be understood.

Disturbances of the function of the bladder are not exactly rare during the first days; there may be complete paralysis of the bladder, with retention of urine, but more frequently there are involuntary evacuations through incontinence of the bladder (and the same thing also holds true with regard to evacuation of the bowels). But these manifestations always disappear completely again within three to eight days. Not seldom, however, especially in little children, a slight weakness of the bladder, with occasional incontinence, remains for a time. But this disturbance, too, is likely to disappear within a couple of weeks or months, leaving no trace behind, and in the later stages of the disease bladder disturbances certainly belong to the rarest exceptions.

The occurrence of bladder troubles is sufficiently accounted for by the occasional somewhat greater extension of the anatomical lesion within the gray substance.

The *sexual functions* probably never suffer any disturbance, or at least only quite exceptionally and rather indirectly (through muscular paralysis, deformities, etc.). They generally remain quite intact, both in men and women.

The same thing is true with regard to the *functions* of the *special senses*, the *psychical* and other *cerebral functions.*

The *vegetative functions* are also usually carried on quite undisturbed. The appetite and digestion are good, bowels sometimes rather sluggish. The general nutrition of the body is usually admirable, if some other circumstance does not incidentally disturb it.

b. Poliomyelitis Anterior Acuta in the Adult.

When this malady attacks adults, we have essentially the same picture of disease presented to us as in children. It is

merely modified, in a non-essential mann
brain of the adult offers somewhat more
disturbances, that the general organism i
to fever, and that the growth of the bone
and the firmness of the joints is greater.

Here, too, the disease begins with a
fever which is generally introduced with
and the extremities, and not rarely witl
tion, a feeling of numbness, etc.). Sever
generally wanting; general convulsions l
served, but *severe headache, dulness, som*
delirium occur. Well-marked *gastric sy*
have been repeatedly met with. In som
great intensity.

Then the *paralysis* is developed, mo
rally in the course of a few hours, pe
rarely not until after a few days. Just as
it is more or less widely spread, comple
cidity of the paralyzed muscles.

Now and then *temporary weakness of*
veloped. Reflex action in the paralyze
ered or extinguished; though in some c
may be retained, at least in those muscle
nently and not completely paralyzed.
ever, need further confirmation, through

An improvement of the general condit
after one or two days, rarely not till th
and then an improvement in the paraly
lows. This, then, either gradually adva
after many weeks and months—to com
paralyzed parts (*temporary paralysis*, ca
merely a partial restitution of the muscle
remain for a long time or permanently
in the case of children, *rapidly progressi*
with evidences of the reaction of deger
becomes lax and withered, the extremitie

But there exists *no trace of disturba*
patient never takes to his bed; the bla

have existed soon subside; the sexual function remains entirely normal; the general nutrition becomes quite good again.

In the further course of the disease the evil consequences of the paralysis and atrophy of muscles inevitably show themselves in the shape of *paralytic* and *myopathic contractures* and the *deformities* resulting therefrom. These, however, never reach the same degree as in children, partly because in adults there is no interference with the growth of bones, and therefore no shortening of limbs, because the greater firmness of the joints and their ligaments in adults prevents more serious deformities of the same, and because adults notice the deformities at their very beginning, and seek, by means of early treatment or careful management, to remove them, or to hinder their further development, which is, of course, not nearly as much the case with children.

The differences between acute spinal paralysis in children and that in adults actually reduce themselves down to this more slight development of the secondary and consecutive alterations in the latter. In all other respects, in the extension of the paralysis and atrophy to single or to many muscles, to one or to all extremities, or to the trunk, in the further course of the disease, in the insignificant influence of the same on the general well-being, etc., there is the most complete correspondence between the two, and it is therefore not necessary to add anything further with regard to the affection in adults.

Course—Duration—Terminations.

There is not much to be added to what has already been said on this subject. The typical and ordinary course of the disease —with its extremely acute beginning and very quick transition to a chronic stage, and with its gradual development of secondary changes—is, in the majority of cases, pretty much the same, and has been described with sufficient minuteness in the preceding pages.

With reference to the *course* of the affection, we will only add a few words on the subject of so-called *temporary spinal paralysis.* The term "temporary paralysis" seems to have been first used by Kennedy; at least he is everywhere cited as authority for the occurrence of temporary paralysis in children, and I think unjustly so, for scarcely any of his cases belong under this general head, and those which do belong under it were not "temporary." At the same time, such temporary forms of acute spinal paralysis do seem to occur, as has been claimed by Duchenne, Volkmann, and others, and established by Frey with several undoubted cases. Neither can we see any reason why the restoration of motility, which in ordinary cases is regularly observed in a part of the muscles, should not in the lighter cases extend to *all* the paralyzed muscles. These temporary forms are distinguished from the permanent ones only in this, that in the course of some weeks, or a few months, *complete* restoration of all movements takes place, although, at the beginning, these cases were by no means to be distinguished from the severer forms; they may even have been characterized by atrophy with loss of faradic irritability. These temporary forms occur in adults as well as in children.

Nothing further need be added with regard to the *duration* of the disease.

The *termination* of the disease seems always to be favorable, so far as its relation to life is concerned. There is no evidence that this disease, of itself, has ever directly induced death. In persons who have been affected with spinal paralysis death results from accidental occurrences, the weakness of age, or any disease imaginable.

Cases of this affection divide themselves, quite simply, into two classes: those in which *complete recovery* takes place—the temporary paralyses just mentioned, and those in which *the recovery remains incomplete*, that is, there is recovery with some remaining defect. This defect is due to permanently paralyzed and atrophied muscles, to the shortening and bending of limbs, etc. Daily experience teaches that this defect, even when it is very great, need not materially interfere with the general well-being, nor the following of an occupation, nor the enjoyment of life. To be sure, there are also miserable cripples enough who have been placed in the saddest predicament for their entire lives by means of acute spinal paralysis, even though they may still enjoy a good appetite and a clear head.

We would here again briefly call attention to one point. It would be well worthy of further attention to investigate whether persons who have once gone through with an attack of acute spinal paralysis do not show a greater disposition to diseases of the spinal cord, so that in later life they are attacked with other spinal affections. I have stated above that this has occurred but seldom in my own experience and that of others—so seldom, in comparison with the absolute frequency of the spinal paralysis of children in general, that I am rather inclined *not* to believe in any such causal relation. Still, this experience may prove to be deceptive, and, on closer investigation, the facts may turn out differently.

Diagnosis.

With the degree of knowledge at our disposal to-day, and in view of the great uniformity of the picture of the disease and its course, it is certainly quite easy to recognize acute poliomyelitis anterior. There will be no difficulty in making the diagnosis where the characteristic grouping of symptoms is present; where a sudden febrile affection, not unfrequently accompanied by severe cerebral manifestations, is quickly followed by a wide-spread paralysis of a high grade, with complete relaxation of the limbs; where early improvement of the general condition, and not long after partial recovery from the paralysis follows, while the more severely paralyzed muscles lose their faradic irritability and fall victims to rapid atrophy; where reflex action is completely extinguished, but cutaneous sensibility and the functions of the bladder and rectum are entirely preserved. Under these circumstances it will not be necessary to wait for secondary contractures and deformities, for the arrest of the growth of limbs, etc., to confirm the diagnosis.

But, even in the lightest, and what might be called fragmentary cases, it will, as a rule, be possible to make a diagnosis with greater or less certainty by means of a thorough examination of the nerves and muscles, careful regard to the development of the disease, and all other circumstances relating thereto. During its latest stages, likewise, the disease may still be recognized by its sequels, and, unless all points in the history (to show the method of its development are wanti nounced upon with certainty. Here, however, imagine the possibility of confounding it with other kindred forms of disease.

We must, however, as a matter of course, b place in this category every instance of spinal] in childhood, even if accompanied with atrophy still too often done. But such a stretching of t is included under "spinal paralysis of childhoo able at the present day, and it must be positive in the diagnosis the *characteristic* signs of th hered to.

Acute poliomyelitis anterior must be disting following forms of disease:

From *acute, central, or transverse myelitis.* tinction will not generally be difficult, on accoun ances of sensibility and of the bladder and re almost invariably present in these diseases; on occurrence of bed-sores; on account of the fa usually increased reflex action and usually no principal marks of distinction are the disturbanc and of the bladder, also the course of the affectic

Hæmatomyelia, or hemorrhage into the gray present great similarity to acute spinal paralysi the rapid development of the paralysis, the subs and reaction of degeneration, the absence of re But the non-febrile beginning of the former, the apoplectiform appearance of the paralysis, the a disturbances of sensibility, the paralysis of the bed-sores, etc., insure the diagnosis of hemorrha

Myelitis from compression—such, for instan leads to rapidly developed paraplegia in Pott's d be confounded with poliomyelitis anterior by inattention, or in quite exceptional cases. The sensibility, the heightening of reflex excitabilit of the bladder, the spasmodic manifestations i tremities, the absence of atrophy and the reacti tion, the presence of affections of the vertebral column, of lively pain, of disturbances of the general health, etc., are enough to determine the presence of this form of disease.

The *cerebral hemiplegia*, which is not rare in children and quite frequent in adults (especially what is called "hemiplegia spastica infantilis"), is to be distinguished from our disease by the more or less complete demonstration of the following symptoms: headache, dizziness, affection of the cerebral and spinal nerves, strabismus, a stupid expression of countenance, paralysis with tension of the muscles and spasmodic contractures, increased reflex excitability of the tendons, no atrophy or but a very slight degree of the same, the preservation of electrical irritability, involuntary additional movements on voluntary motion of the sound side of the body, stiffness and extension of the joints, the bones not at all or but little shortened, more or less considerable disturbances of sensibility, no scoliosis, etc. These signs are also sufficient for a differential diagnosis, even when such a cerebral affection is bilateral.

Progressive muscular atrophy is to be distinguished from acute spinal paralysis simply by its chronic development, the progressive course of the disease, and the lesser prominence of the paralysis; furthermore, by the retention of electrical irritability, the hereditary relations of the disease, etc.

Pseudo-hypertrophy of the muscles is likewise to be recognized by the slow development of the disease, the absence of severe paralysis, the increase in the volume of the muscles, and the absence of any atrophy.

The *spasmodic spinal paralysis* of children, two cases of which I have reported,[1] is very easily to be distinguished by the slow, insidious and unnoticed development of paresis (which seldom goes on to complete paralysis), by the muscular tension and contractions, the increased irritability of the tendons, and the absence of atrophy and the reaction of degeneration.

The distinction between our disease and *the paralysis of children following delivery*,[2] which, at a certain stage, may have some resemblance to the spinal paralysis of children, of limited localization, is generally determined by the history of the case and when this is not true, by the characteristic localization, as well as by the disturbances of sensibility that are present; furthermore, by the absence of a febrile initial stage, the occurrence of the trouble at an early age, etc.

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[1] Virchow's Archiv. Bd. 70. Heft 3. 1877.

[2] See this Cyclopædia. Vol. XI. (*Erb*, Diseases of the Peripheral Nerves), p. 561.

The *peripheral paralyses of single groups of muscles and nerves* through pressure, faulty position, tight bandaging, etc., such as not rarely occur in children, will be correctly pronounced on by demonstration of the cause, by the absence of the characteristic initial stage, by the strict localization of the trouble in a definite peripheral nerve-domain, by the presence of disturbances of sensibility, by the rapid, favorable result, etc.

For the distinction between this and *subacute* and *chronic poliomyelitis*, see the following section.

Prognosis.

Acute atrophic spinal paralysis does not appear ever directly to threaten life; its prognosis is therefore absolutely favorable, so far as life is concerned. The first onset, it is true, often looks dangerous, and it does not appear exactly impossible that death may occasionally follow during the initial stage. It is a question which as yet remains undecided, whether some cases of death from "convulsions" do not belong in this category. This can only be decided by a very careful and intelligent examination of the spinal cord in such cases. I was forcibly struck by the fact that in von Heine's histories of his patients it repeatedly appears that one or another child in the same family had died of convulsions.

The prognosis with reference to complete recovery is quite different. Here it may be pronounced as almost absolutely unfavorable. The "temporary" forms of the disease are so rare that they can hardly be taken into consideration; in fact, the best we can do, almost always, is to make a prognosis of *recovery with greater or lesser remaining defects.* The parts that do not regain their mobility in the first two or three months, or within the first six months, seldom do so at a later period.

There is then little to be expected in the way of direct attempts at recovery, although slight and partial improvements, even at later periods, are not exactly rare.

At the same time the usefulness of the extremities may still be materially improved through appropriate orthopædic operations, gymnastics, electrical treatment, etc., and thus the magnitude of the defect be rendered less noticeable by the patient.

The prognosis in this respect depends upon the grade and extent of the paralysis, upon the preservation and the functional capacity of certain muscles and groups of muscles, upon the extent of the atrophy and the amount of deformity already present, upon the age of the patient and of the disease at the beginning of treatment, upon the intelligence and perseverance of the patient, and more of the same kind. A rich experience will of course sharpen one's judgment with regard to the value of these several conditions.

Therapeutics.

The treatment of acute spinal paralysis naturally falls into two divisions: the treatment of the *acute inflammatory attack*, and that of its *sequels* and *results* (the paralysis, atrophy, contractures, and deformities).

The fulfilment of the first task is rendered difficult by the fact that the endeavors of the physician often come too late, or that his diagnosis remains in doubt until the paralysis is fully developed.

In all cases, however, which come under treatment in good time, the measures which would be applicable in acute myelitis are to be adopted with all energy—of course, in a manner fitting the circumstances, and especially with due reference to the tender age of many of the patients. Local abstraction of blood, therefore (leeches, cups, etc.), is to be employed, first of all, at the parts especially affected (the lumbar and cervical enlargements); cathartics (calomel, infusion of senna, etc.); inunction of mercurial ointment to the back; the use of ergotine, belladonna, or iodide of potassium in appropriate doses; derivatives (strips of blister plaster the length of the spine, painting with tincture of iodine, etc.); and, in appropriate cases, the application of ice, o the employment of Priessnitz's cold wraps about the trunk. I cases of severe brain-symptoms, Kussmaul recommends a tepi bath, with cold affusion to the head. Still, we need not b immediately driven to more active interference by apparentl threatening manifestations, as experience teaches that there i hardly ever any real danger during the initial stage. But if w should succeed, by energetic treatment, in suppressing the myelitis at its outset and thus preserving a portion of the nervou elements from positive destruction, it would be a great poin gained.

In the second stage of the disease it must likewise be ou main endeavor to induce a favorable change in the diseased focu itself within the spinal cord, to favor the resolution of the inflammatory and degenerative changes, so that whatever of nerve-elements, fibres and cells, may still be capable of being saved, shal be saved and again put in condition to perform their function Those ganglion-cells which have once been totally destroyed cannot be reproduced, but probably cells and fibres which are in th act of degeneration may be preserved from further destructio and gradually brought back again to the performance of thei functions. It is only on this supposition that there can be an sense in the further symptomatic treatment of peripheral nerve and muscles. It is hard, indeed, to determine how far the powe of our curative measures reaches towards the fulfilment of thi indication; up to the present time not much can be said in prais thereof.

The *galvanic current* may undoubtedly be regarded as one o the principal means of influencing the diseased area within th spinal cord; and it is the catalytic action of the current that i to be tried. What we want, therefore, is a stabile action of th current upon those portions of the spinal cord which are chiefl diseased. Our best plan will be to place a large electrode, whic will cover the entire diseased area, over the corresponding portion of the back (over the lumbar or cervical enlargement), an to apply the other to the anterior surface of the trunk, and firs to let the anode, then the cathode act for from one to two minutes at a time, with a moderately strong current. The earlie this treatment is begun, the better; during the later stages little is to be expected from it, although, according to the well-established experience of some, a favorable effect is by no means impossible. The treatment must be continued for a long time, or must be repeated, at intervals, for several years.

It must be admitted that, as yet, this treatment has been followed by no specially brilliant results; this is perhaps partly due to the fact that these cases almost always come under treatment far too late, and that physicians have often contented themselves with the peripheral application of the current to the paralyzed muscles, instead of making the diseased area within the cord itself the main object of galvanic treatment, as should always be done. It is also quite evident that the faradic current is not the one to be used in these cases. According to my own experience, which relates almost exclusively to old cases and later stages, I can report no brilliant results; still, I have often seen improvement set in, which I was obliged unquestionably to ascribe to the treatment, so that I cannot concur in the unfavorable verdict of Volkmann on electrical treatment. This verdict has also already been corrected by Hitzig and Juergensen. To be sure, we may not expect much from inappropriate, inadequate, or purely peripheral electrical treatment, and under all circumstances the attainment of any considerable result requires unusual patience and perseverance on the part of the physician as well as the patient.

The same end—healing and resolution of the changes within the spinal cord—may be sought to be reached through the *general stimulation of tissue-changes and of nutrition.* On this ground something is to be expected from the use of baths (thermal springs, saline springs, thermal saline springs rich in gas, animal baths, pine-needle baths, cold-water cures, sea-baths, etc.). No more precise indications for the selection of the kind of baths can yet be given. For children, I should first use saline springs and saline thermal springs, afterwards light cold-water treatment; and for somewhat larger children, also sea-baths (and sea-air). For adults, I would recommend gaseous saline thermal springs, and then the energetic cold-water cure.

On the same principle, a *strong and abundant diet* will be of benefit, with prolonged sojourn in the open air—the air of mountains and woods—and the use of *cod-liver oil.* It is a question whether *rubbing the skin with spirituous or other applications* may have a similar though feebler effect, by exciting the activity of the skin and aiding the circulation; at all events, it may be tried.

As a matter of course, all sorts of internal remedies have been tried—iodide of potassium, iodide of iron, nitrate of silver, etc.—all, at best, with doubtful results. Strychnine has been warmly recommended by many for the second stage; Heine and Sinkler have found it alike inefficacious when used internally or externally. In view of the much surer and safer action of electricity, its use may well be ignored. Hammond recommends ergot to be given as early as possible, and in full doses.

Finally, *symptomatic treatment* is of the highest importance. This is to be directed, first, against the paralysis and atrophy of the muscles. The principal means to be used is electricity. In addition to the direct galvanic treatment of the spinal cord itself, the systematic peripheral treatment of the nerves and muscles is also to be carried on. Naturally, the galvanic current is the one to be used first, on account of the reaction of degeneration of most of the muscles. Still, Duchenne has arrived at quite noteworthy results even with the faradic current; this is especially applicable to those nerves and muscles which have but slightly lost or entirely retained their faradic irritability. But these muscles, naturally, also offer the best chances for galvanic treatment. The method of treatment is simple: the labile application of the cathode over all the paralyzed muscles and nerves, while the anode is best placed upon the vertebral column at the height of the main lesion. For very much atrophied and but slightly irritable muscles, changes in the current are to be recommended in which both electrodes are to be applied to the muscle (one upon the nerve-trunk belonging to it). The strength of the current should be such as to produce distinct twitchings and lively redness of the skin. The faradic application is to be made with moist electrodes, and with a pretty strong current. Treatment must always be continued for a very long time—months and years—with longer or shorter intervals. By this means some slight progress is generally attained.

In support of the electrical treatment, *massage and kneading of the muscles*, together with appropriate *gymnastic exercises*, may be undertaken. *The application of warmth* (warm-water compresses, sand-bags, etc.) has also been recommended to further the blood-supply and nutrition; and for this purpose, too, some have advocated *friction* of the limbs *with irritating substances* (oil of mustard, tincture of cantharides, strong liquor ammoniæ, extract of nux vomica, etc.).

But the main portion of the treatment generally, and in old cases always, falls within the domain of *orthopædic surgery*. It is not our province here to enter into details on the difficult and manifold indications which arise in this connection; we refer our readers, for that, to treatises on surgery and orthopædics. We will only allow ourselves a few brief observations on the main principles of the orthopædic treatment.

The main point, and the most important task, which devolves precisely upon the practitioner who has these cases under treatment at the beginning, is the *prevention* of contractures and deformities; when these are once developed and have grown old, they are a more appropriate object for the specialist in orthopædics and for orthopædic institutions. The physician may render considerable service in this respect, if he keeps in mind the purely mechanical methods of origin of the majority of contractures and deformities, and early enough counteracts these mechanical causes. This is the main point, and in its accomplishment electricity, gymnastics, light frictions, and baths can serve merely as mild adjuvants.

The essential point consists in watching the position of the limb when at rest, and in guarding the movements and the gait so as to avoid the undue burdening of one side of the body and continuous false positions. In guarding against the occurrence of talipes equinus it is sufficient, according to Volkmann, if, during the earliest stage, while the patient is lying down, the foot be fastened to a light foot-board by means of a flannel bandage, and this be drawn up somewhat towards the leg by a strip of adhesive plaster. On their attempting to walk, children should be put into stout-laced boots, with a steel shank on the outer or inner side of the boot-leg, or with the sole slightly thicker on the one side or the other; in this way the develoj ment of talipes varus or valgus may be counteracted. The fo mation of talipes calcaneus may be combated by supplying th defective action of the posterior muscles of the leg by a stron bridle of rubber (the best is a rubber ring), which passes from th heel to a trough-like fixture that is applied to the leg just belo the knee, and that is held firmly in place by a side-bar fastene to the shoe. Indeed, the lost muscular power, wherever it occur may be replaced in various ways by means of elastic tugs an the like.

According to the seat and extent of the paralysis, accordin to the kind and magnitude of the deformity, the most varie machines and supporting apparatuses are to be applied, in th construction of which orthopædic surgery has accomplished great deal, and which often secure to the patient a very conside able use of his limbs.

In the more severe deformities tenotomy and forcible mea of correction may come into play. According to Volkman patients should, as far as possible, avoid the use of crutches an similar supports, especially at the beginning of their ailment.

16. *Subacute and Chronic Inflammation of the Gray Anteri Horns.—Poliomyelitis anterior, subacuta et chronica.- Chronic atrophic spinal paralysis.—Paralysie générale sp nale antérieure subaiguë* (*Duchenne*).

Duchenne (*de Boulogne*), Recherches électrophysiol., patholog. et thérap. Comp rend. de l'Acad. d. Sc. 1849—De l'électrisat. localisée. 1855. 3. édit. 1872. 459.—*Nesemann*, Heilung eines bis zur vollständ. Lähmung aller Extremitäte vorgeschritt. Falles von progress. Muskelatrophie u. s. w. Berl. klin. Woch 1868. Nr. 37.—*Poché*, Quelques considér. sur les amyotroph. d'origine spinal Thèse. Paris, 1874.—*Frey*, Fall von subacut. Lähmung Erwachsener. Berl klin. Woch. 1874. Nr. 44, 45.—*Erb*, Ueb. acut. Spinallähm. bei Erwachs. und über verwandte spinale Erkrankung. Arch. f. Psych. u. Nervenkrankh. V Beob. 7. 1875.—*Cornil* et *Lépine*, Cas de paralys. génér. spin. antér. subaiguë suivi d'autopsie. Gaz. méd. de Par. 1875. Nr. 11.—*Lemoine*, Paralys. de l'adulte Guérison. Lyon médic. 1875. Nr. 15.—*Lincoln*, Case of Spinal Paralysis in an Adult. Bost. Med. Surg. Journ. 1875. March 25.—*S. G. Webber*, Contrib. t the Study of Myelitis. Transact. Americ. Neurol. Assoc. for 1875. Vol. I. p

55.—*Goltdammer*, Ueb. einige Fälle von subacuter Spinalparalyse. Berl. klin. Woch. 1876. Nr. 26.—*Hammond*, Diseases of the Nerv. Syst. 6. edit. 1876.—*Klose*, Zur Lehre von der Paralys. spinal. anter. subacut. Diss. Breslau, 1876. —*Bernhardt*, Beitr. zur Lehre von der acut. atroph. Lähmung Erwachsener. Arch. f. Psych. u. Nervenkrankh. VII. S. 318. 1877.

History.

It was Duchenne who, first in the year 1849, and then again in 1853, described a peculiar form of spinal disease, characterized by motor paralysis, progressing more rapidly or more slowly, accompanied with atrophy in bulk of the muscles, and loss of their faradic excitability, without any other manifestations.

This statement attracted but little attention until Duchenne, in the third edition of his "Electrization localisée" (1872), again and more emphatically pointed out the existence of this form of disease, and gave a detailed description of the same. Influenced in part by a hypothetical belief with regard to the anatomical basis of the disease—which he believed to consist in chronic degeneration of the gray anterior horns—he designated it "Paralysie générale spinale antérieure subaiguë."

Since that time the existence of the disease has been universally recognized as a clinical fact, and single instances of it have been described by various authorities (Poché, Frey, Erb, Webber, Cornil et Lépine, Klose, Goltdammer, Bernhardt, and others).

The only point on which there is still any doubt is with reference to the anatomical basis of the disease, although the single reports of post-mortems thus far made by Cornil et Lépine and by Webber, would seem strongly to confirm the hypothesis first set up by Duchenne. Further observations on this subject are desirable.

Definition.

Clinically considered, the disease presents itself as a motor *paralysis*, usually developed without fever, with but slight general disturbance and insignificant disturbances of sensibility.

The paralysis more or less rapidly seizes the entire lower extremities—generally in the course of a few days, or at the most a few weeks—and soon extends to the upper extremities also (much more rarely showing the opposite order of development and beginning in the upper extremities); it is associated with *complete flaccidity of the muscles and loss of their reflex excitability*, and is followed by *rapidly progressive atrophy in the bulk of the paralyzed muscles*, with the well-marked *reaction of degeneration.*

The disease has certainly, as a rule, an ascending course, though it is by no means always progressive; its development generally comes to a stand-still sooner or later, this arrest introducing a gradual retrogression of the disturbances, which may lead to more or less complete recovery.

The *anatomical limits* of the disease cannot, as yet, be declared with absolute certainty; but, according to all that we know, there is every probability of its being located in the gray anterior horns. The two post-mortem examinations thus far made virtually confirm this, and thus, for the present, we may designate the disease as a *subacute or chronic inflammation or degeneration of the gray anterior horns*, with extensive disappearance and atrophy of the large multipolar ganglion-cells.

Etiology.

The causes of the disease under consideration are as yet exceedingly obscure. Nothing is known of any definite *predisposition* thereto, and no hereditary influences have as yet been demonstrated. All the cases observed thus far have been in adults, and, indeed, between the ages of thirty and fifty, as is also the case with the majority of other chronic spinal diseases.

Under the head of *exciting causes*, traumatic injuries have been cited—a fall upon the hip or back; furthermore, gross exposure to taking cold, damp dwellings, and, in the case of Klose, free indulgence in beer and excesses in venery. All these are mentioned as possible etiological conditions. In the majority of cases, however, no definite cause for the disease can be demonstrated.

We must, however, here briefly call attention to one etiological factor which is, as yet, of hypothetical, though perhaps of very great significance: that is, *chronic lead-poisoning.* In speaking of the pathogenesis of lead paralysis,[1] which is such a peculiarly characteristic affection, I have sought to show that its origin is probably spinal instead of peripheral, and E. Remak[2] has established this more accurately and in a more detailed manner by the sifting and critical examination of a larger amount of material. He comes to the conclusion that quite circumscribed alterations in the gray anterior horns probably lie at the foundation of lead paralysis. These alterations might well be of a degenerative or chronic inflammatory nature, but are, as a rule, capable of resolution. Bernhardt has also recently given in his adherence to this view. In fact, the whole state of things in lead paralysis, with reference to mobility, atrophy, electrical relations, the absence of disturbances of sensibility, etc., is so entirely analogous to that in poliomyelitis anterior chronica, that we are almost forced to the theory of changes in the anterior gray horns in lead paralysis. On the other hand, again, I recently saw two cases of paralysis of the upper extremities, which, in all their details, were so completely analogous to lead paralysis that only the entire lack of any sort of evidence of lead-poisoning was sufficient to determine me to give up this diagnosis, and accept that of a chronic circumscribed poliomyelitis anterior. Thus, everything crowds us at least into regarding the localization of lead paralysis and of chronic anterior poliomyelitis as the same. It is left to further investigations to determine whether the saturnine alterations in the gray anterior horns are the same in *kind* as the others—whether, therefore, the lead-poisoning calls forth an *inflammatory* affection. It would be well, at all events, to keep this question in view in the future.

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[1] *Erb*, Diseases of the Peripheral Nerves. This Cyclopædia. Vol. XI.

[2] Zur Pathogenese der Bleilähmungen. Arch. f. Psych. und Nervenkrankh. Vol. VI. p. 1. 1875.

Pathological Anatomy.

Up to the present time there are but two reports of post-mortems before us, which certainly do not fulfil all the requirements of a vigorous critique. We here communicate them in brief.

In one case, which terminated in death at the end of four years, Cornil and Lépine found softening of the lowest division of the spinal cord. The principal change was in the gray anterior horns, consisting of chronic inflammation, thickening of the vessels, an abundance of fatty, granular cells, the perivascular spaces filled with blood, disappearance of a great part of the ganglion-cells, increase of the cells of the neuroglia. In the cervical portion there was a sclerosis of the antero-lateral column entirely surrounding the anterior gray horn, and being limited farther down to the posterior divisions of the lateral column (secondary degeneration ?). The anterior roots were atrophied; in the muscles the usual fatty atrophic changes were found.

In Webber's case, which had lasted eleven months, he found degenerative atrophy of the muscles, the anterior roots in a high state of degeneration, the posterior roots but very slightly degenerated. In the *gray substance* there was here and there an exudation and an increase of nuclei around the vessels ; disappearance, atrophy, and degeneration of the large ganglion-cells in the anterior horns ; but slight changes in the ganglia of Clarke's columns and of the posterior horns ; in the white columns a part of the nerve-fibres were degenerated, the neuroglia hardly changed. The same alterations which Webber ascribes to a pre-eminently parenchymatous inflammation were found to extend as far up as into the medulla oblongata.

From these two not altogether coinciding reports, this much, at least, would appear evident : that even if the lesion is not entirely confined to the gray anterior horns, it is at least most pronounced in these ; and that what here especially attracts attention, in addition to the chronic inflammatory changes, is the degenerative atrophy of the large, multipolar ganglion-cells. In the anterior roots and in the muscles those changes are to be found which have already repeatedly been described as being characteristic of degenerative atrophy of these structures.

Pathology of Poliomyelitis anterior chronica.

Symptoms.

It will suffice for all practical purposes to give a somewhat minute *general picture* of this, on the whole, rare affection. A more detailed pathogenetic discussion of its symptoms would bring to light the same occurrences as constitute the principal manifestations of the acute form.

The disease may be developed with very varying degrees of rapidity; sometimes in a more subacute manner, developing a widespread paralysis within a few days or weeks, but generally pursuing a more chronic, insidious course, so that its development may extend over years.

Accordingly, light indications of fever, gastric disturbances and dyspeptic manifestations, headache, and the like, may be present or may be wanting at the beginning of the disease. On the other hand, we shall rarely fail to encounter all kinds of *paræsthesia* and *light manifestations of sensory irritation* (drawing and shooting pains in the back and limbs, backache, etc.) among the precursors of the disease. It is also common to find that for a long time such persons have suffered from *great weariness* and diminished endurance in the limbs.

The actual *beginning of the disease* is generally marked by a *distinct motor weakness* in the lower extremities, or in but one of these, or at least more prominently in the one. This weakness more or less rapidly grows distinct; it increases in the course of days or weeks to *pronounced paresis*, so that the patient breaks down on going upstairs, and soon finds his walks reduced to a minimum, or is confined to his bed. During this entire development there is never any tremor, never any ataxy in the limbs.

On examining such patients, their movements are found to be sluggish, awkward, weak, and paretic; the movements of the knee and ankle-joint are usually more severely interfered with than those of the hip-joint. Sooner or later this paresis increases to complete *paralysis* of single muscles and groups of muscles, or of the entire extremity. The rapidity with which this takes place varies exceedingly in different cases; sometimes it occurs within a few days, sometimes not till after the lapse of many months, or even after years. Then the muscles are *completely flaccid* and soft, the limbs easily movable in all directions; at first there is no sign of any contracture.

Very soon a progressive *atrophy in bulk* takes place in the paralyzed muscles; the calves of the legs turn into lax, flabby sacks, the muscles of the thigh and gluteal region grow thin and flaccid, and the tape-measure shows a diminution in the volume of the muscles from week to week. This atrophy may advance till it causes a skeleton-like emaciation of the limbs, amounting to almost entire disappearance of the muscles. In its earlier stages it is generally accompanied with more or less frequent and active fibrillar twitchings of the muscles.

The feet are cold and easily grow cyanotic. *Sensibility of the skin* is usually completely intact in all directions; it is but rarely that this is slightly dulled and that patients complain somewhat of insensibility and numbness.

Reflex action in the paralyzed muscles is *completely extinguished;* neither through the skin nor through the tendons can the slightest reflex twitching be produced.

It is usually not long before the affection also extends *to the upper extremities;* these grow awkward, feeble, heavy, gradually paretic, and at last completely paralyzed. Here, too, the trouble extends more prominently or earlier to individual groups of muscles; sometimes the extensor group of the forearm is earlier and more severely paralyzed than the rest, sometimes the flexors and the little muscles of the hand are more prominently affected. In general the fingers and hands are more severely paralyzed than the forearm and shoulder. The hands assume a correspondingly characteristic position, the arms lie lax and immovable as they are placed, no trace of any contracture being demonstrable.

Reflex action is completely extinguished in the paralyzed muscles. Sensibility in the upper extremities, too, so far as objective examination can prove, seems to be entirely normal, or diminished to an entirely insignificant degree. Subjectively, patients sometimes complain of numbness in the fingers, of paræsthesia in the domain of the ulnar nerve, etc.

Here, too, rapidly progressive atrophy ensues, uniformly distributed over the paralyzed muscles, which may lead to the highest degrees of emaciation, especially in the hands and forearms.

In some instances the muscles of the back and abdomen also share in the paralysis: the patients can no longer sit up; expiration, coughing, sneezing, defecation, etc., are rendered difficult.

There is generally an entire absence of any disturbances of *the bladder*, *the rectum*, *or the genital* organs; the functions in question are carried on undisturbed and in quite a normal manner; the exceptions to this are rare.

The nutrition of the skin does not suffer in the least; no bedsores occur.

The vegetative functions are also all of them normally performed. The appetite and digestion, which may, at the worst, have been slightly and temporarily impaired at first, are good; sleep is natural; the general nutrition and well-being are usually quite undisturbed.

No symptoms at all on the part of the brain or the cerebral nerves appear, at least during the earlier stages of the disease. The initial headache ordinarily soon disappears.

Examination of the paralyzed and atrophied muscles by electricity gives quite the same results as in acute anterior poliomyelitis, only modified to a slight degree to correspond with the much slower development of the malady.

Here, too, Duchenne showed a diminution of faradic excitability occurring from the beginning, and advancing more or less rapidly to complete loss of the same.

Here, too, according to my own experience and that of others (Bernhardt), this is nothing more than one of the manifestations of the *reaction of degeneration.* I have been able to prove this electrical disturbance in all the more recent cases that have come under my notice (five in all), and indeed in its most pronounced form. The motor nerves were entirely unexcitable to both the faradic and the galvanic current; the muscles did not respond to faradization, their galvanic excitability being retained, and during the first weeks and months of the disease being slightly heightened (see the case reported below), afterwards more or less diminished, but always changed in quality (AnSZ > KaSZ; twitching sluggish, tonic); their mechanical irritability was at first heightened.

At the stage in which we generally first see these cases, galvanic excitability is generally already much lowered, but usually still permits of the very distinct recognition of the qualitative anomaly. With the cure of the disease the electrical excitability returns but very slowly and gradually to its normal standard.

We have now become familiar with the occurrence of the "reaction of degeneration" in various diseases of the spinal cord, and it would seem to be time that we should enter briefly upon the relations of the same, in order to throw some light upon its diagnostic significance and to indicate the important points in general pathology which result from the connection of this with other already recognized facts.

We have found that the reaction of degeneration appears in its full development in *acute anterior poliomyelitis* in the form of complete loss of faradic and galvanic excitability of the motor nerves, loss of faradic excitability of the muscles, but heightening and qualitative changes in the galvanic excitability of the muscles. We have, therefore, in this form of disease, complete paralysis, a high grade of atrophy, the motor nerves and muscles involved in degenerative atrophy.

Quite the same thing is found—only somewhat milder in degree, corresponding to its more chronic course—in *chronic anterior poliomyelitis:* the complete reaction of degeneration, the electrical excitability of the nerves entirely abolished, etc.; here, therefore, likewise entirely the same condition, *viz.*, paralysis, atrophy, the motor nerves and muscles degenerated.

Quite a different state of things exists in *amyotrophic lateral sclerosis.* A case which I accurately examined of late showed, in the upper extremities, the faradic and galvanic excitability of the motor nerves entirely preserved (at all events not materially diminished); in the muscles an elevation and qualitative change of galvanic excitability (reaction of degeneration) on direct irritation, while on indirect irritation the muscles reacted normally (twitching like lightning, KaSZ > AnSZ); the muscles also responded to the faradic current.[1] At the same time there was a high degree of atrophy and complete paralysis of the muscles. Here we therefore found paralysis, atrophy, the motor nerves *not* degenerated, the muscles degenerated.

In *progressive muscular atrophy* (typical form) it is somewhat different again.

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[1] The same condition, therefore, exists which is characteristic of the so-called middle form of facial paralysis and some other peripheral forms of paralysis. Compare this Cyclopædia, Vol. XI.

In a part of the muscles attacked, the excitability, through the nerve, is preserved or appears to be simply lowered; in the muscles themselves faradic excitability is preserved, but galvanic excitability is preserved with the characteristics of the reaction of inflammation (though generally with already very much lowered excitability). In another portion of the muscles—in those that are *very* highly atrophied—faradic excitability is abolished, the nerve is entirely unexcitable, and the muscle reacts only on a very heavy galvanic current with sluggish twitching, AnSZ>KaSZ. In those muscles, however, which are but slightly affected and which were attacked at the beginning, electrical excitability appears still to be quite normal on direct and indirect irritation. At least so I have found it, since paying attention thereto, in *all* the cases carefully examined by me. If Bernhardt (l. c.) recently doubts the occurrence of the reaction of degeneration, this is to be excused on the ground that, in progressive muscular atrophy, at all events, the reaction of degeneration is very hard to demonstrate, and may easily be overlooked in a not very careful search. There are various reasons for this. One is the very slow development of the disease, so that the characteristic increase of excitability subsides and the qualitative changes alone remain; another is the preservation of the excitability of the nerves, so that the normal neuro-muscular twitchings mark the qualitative alterations. A still stronger reason, however, is the fact that the degeneration of the fibres within a muscle takes place in a scattered manner; among the degenerated fibres there still always remain a certain number of healthy and still excitable fibres, which respond to irritation, and thus mark the existence of the reaction of degeneration. The more slowly the process advances, the lower sinks the excitability of the degenerating fibres, and the increasing strength of the current required to produce an effect may for a long time compensate for the numerical diminution of healthy fibres; so that the relation of things long remains obscure. It will, therefore, only be possible to demonstrate the reaction of degeneration in its earlier stages in those muscles which degenerate pretty rapidly, and in which larger numbers of degenerated fibres are to be found; the later stages of this reaction (with great diminution of galvanic excitability) can only be shown in those muscles which have already attained a high grade of degeneration. This naturally varies in each individual case. Consequently, the reaction of degeneration can always only be demonstrated in single muscles, sometimes more and sometimes less distinctly. I have thus far always found it in this way.

In progressive muscular atrophy, therefore, the reaction of degeneration exists with retained excitability of the motor tracts (at least for a long time); but, in contrast to amyotrophic lateral sclerosis, *no* paralysis here exists until the muscles are almost entirely degenerated. Here, therefore, the manifestations are grouped as follows: *no* paralysis, but atrophy; the motor fibres *not* degenerated, the muscles degenerated.

I recently found quite an analogous state of things in a well-pronounced case of *bulbar paralysis*. In the muscles of the chin and lips, and even of the tongue, the excitability of the nerves was found retained and almost normal; in the muscles themselves, however, the well-marked reaction of degeneration on direct irritation.

I had expected this condition on account of the analogy of the disease with progressive muscular atrophy.

Quite similar variations are also to be met with in *other forms of paralysis.* Among these may be mentioned the occurrence of the well-marked reaction of degeneration in *severe peripheral traumatic and rheumatic paralysis;* the occurrence of the "middle form" in *slight rheumatic and traumatic paralysis* (of the facial, radial, etc.[1]); finally, the extraordinary fact, in a case of *lead-poisoning* made known by me,[2] where the reaction of degeneration was present in a muscle that was *not* paralyzed, whereas ordinarily in lead-poisoning the muscles act just as in chronic anterior poliomyelitis.

It is difficult to form a plausible conception of the course and connection of the various trophic and motor tracts which might, even to some degree, enable us to comprehend these various facts. Some of the conclusions to be drawn from the facts briefly collected in what has been said above may even now be indicated.

If it had needed any confirmation, all the facts would have unanimously confirmed the doctrine that the occurrence of the reaction of degeneration is always and everywhere associated with the existence of certain histological alterations in nerves and muscles which we comprehend under the name of degenerative atrophy.[3]

But it would furthermore also appear *that the motor tracts, within the central organ, to some degree run their course separately from the trophic apparatuses and tracts,* as there are central paralyses *with* atrophy (poliomyelitis anterior) and *without* atrophy (spastic spinal paralysis), and likewise central atrophies which exist for a long time *without* actual paralysis (bulbar paralysis, progressive muscular atrophy).

We can, furthermore, hardly escape the conclusion that the trophic influences for motor *nerves* are somehow separated in space from those for the *muscles;* that, accordingly, *different trophic central apparatuses and tracts may perhaps exist for the nerves and the muscles.* This appears from the fact that the muscles may degenerate *alone,* without the nerves taking part in the degeneration. (So in amyotrophic lateral sclerosis, in bulbar paralysis, in progressive muscular atrophy, in the middle form of paralysis of the facial and radial nerves, in the case of lead-poisoning described by me.) At all events, therefore, these tracts cannot be completely identical; they must present some differences in their localization, or perhaps only in their resistance to influences that cause disease.

We can, even though it may be with some difficulty, form a hypothetical idea of the existence and position of the various apparatuses and tracts belonging here, within the central organ as well as in the peripheral nerves, whereby we might explain the above-mentioned differences in the different forms of disease. All the facts go to show that these apparatuses lie principally in the gray substance of the anterior horns. But our anatomy will not so soon show us how these are arranged and distributed there. We may, therefore, be permitted, with all due reservations, to draw a hypothetical diagram of this arrangement, according to the postulate which virtually results from the facts above communicated.

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[1] Compare the appropriate sections in Vol. XI. of this Cyclopædia.

[2] *Erb*, Ein Fall von Bleilähmung. Arch. f. Psych. u. Nervenkrankh. Bd. V. p. 445. 1875.

[3] This Cyclopædia, Vol. XI.

In the accompanying Fig. 17, let *a* be the motor conduction from the brain, which undoubtedly lies in the lateral column; let this continue through the ganglionic point *d* (multipolar ganglion-cell ?), into which the reflex tract, *s*, coming from the sensitive sphere, opens through the anterior roots and the peripheral nerves (*a*—*d*—*a*′) to the muscle *m*. Let *b* be the trophic central apparatus for the muscles; the tract *b*—*b*′ represents the trophic conduction tract emanating from this apparatus and somewhere (probably at *d*) uniting with the motor tract. Let *c* be

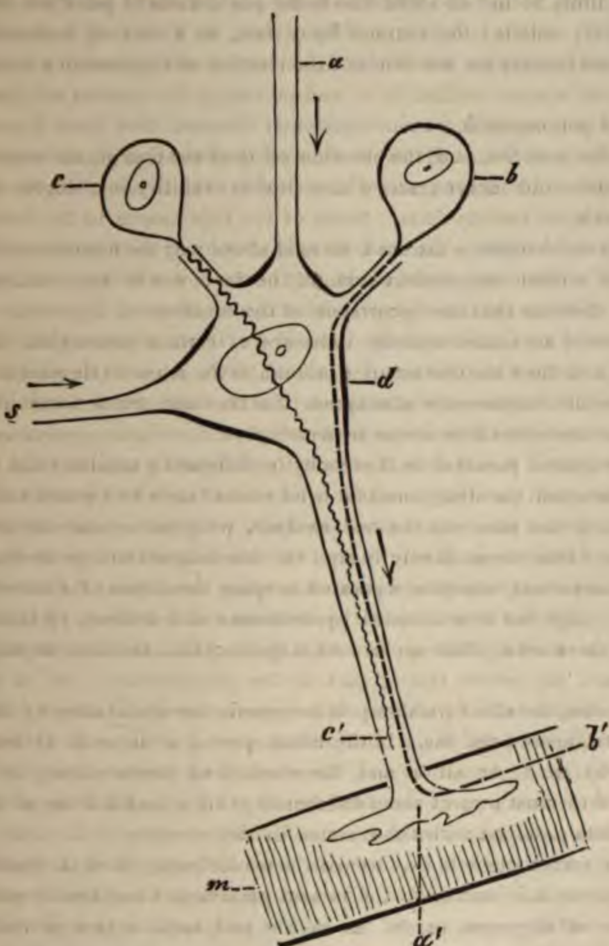

FIG. 17. Diagram of the arrangement and connection of motor and trophic centres and tracts in the spinal cord and in the peripheral motor nerves.

the trophic central apparatus for the motor nerves (which possibly coincides with *d*) and *c—c'* the tract which conducts its trophic influences to the motor fibres.

With this diagram, the special arrangement of which within the spinal cord is of course unknown—and indeed, for the present, a matter of entire indifference—one can form quite a clear idea of the origin of the different forms of disease, provided, at the same time, he has due reference to the phenomena of reflex action.

If the tract *a* alone is interrupted by disease, we have simple paralysis without any degenerative atrophy, and without the reaction of degeneration—*simple lateral sclerosis.*

If *a* and the centre *b* are simultaneously diseased, then there is paralysis with atrophy of the muscles, and the reaction of degeneration in the muscles, but no change of the motor nerves, whose electrical excitability is retained—*amyotrophic lateral sclerosis.*

If the centre *b* alone is diseased, there is atrophy of the muscles, at first without paralysis and without degenerative atrophy of the motor nerves, reflex action being retained—*bulbar paralysis and progressive muscular atrophy.*

If *b*, *c*, and *d* are simultaneously diseased (or, if we suppose that all the tracts pass through *d*, then on disease of *d* alone), there is paralysis with degenerative atrophy, and the reaction of degeneration in nerves and muscles and loss of reflex action—*acute and chronic poliomyelitis anterior.*

If we imagine *b*, *c*, and *d* to represent the different ganglion-cells of the gray anterior horns, and the diagrammatic conduction tracts to represent the different connections of the same among one another, with the lateral columns and the anterior roots, then we shall readily see that this diagram to a great degree harmonizes with the recently accepted views concerning the forms of disease which here occupy us. Even the view adopted by Duchenne and Joffroy, by Hammond and others, that there are separate motor and trophic cells, has found representation in the diagram.

But this diagram also furnishes points towards the elucidation of the different forms of peripheral paralysis. If the conduction *d*, *a'* alone is disturbed, simple paralysis, without degeneration and the reaction of degeneration, will exist (the light form of rheumatic paralysis of the facial); if *d*, *a'* and *b*, *b'* are simultaneously disturbed, then paralysis with the reaction of degeneration in the muscles, but not in the motor-nerves (the middle form of rheumatic paralysis of the facial); finally, if in addition to *d*, *a'* and *b*, *b'*, *c*, *c'* is also paralyzed, then there is paralysis with the reaction of degeneration in the nerves and muscles (severe form of facial paralysis[1]).

But, aside from these more theoretical, and, as yet, entirely hypothetical sides of the question, accurate electrical examination also has its diagnostic importance in such forms of disease. So far as one can judge, up to the present time, with the very scanty material thus far on hand, some not unimportant diagnostic facts would seem to come to light.

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[1] Compare my Essay, cited above—A Case of Lead-Poisoning. L. c. p. 455.

Well-marked, complete reaction of degeneration, with loss of the excitability of the motor-nerves and with elevation, or, at least, without any great degree of diminution of the galvanic excitability of the muscles, is evidence in favor of *poliomyelitis anterior*, both of the acute and chronic form. It is always accompanied with paralysis, disappearance of reflex action, and a high degree of atrophy.

The middle form of the reaction of degeneration, without any material disturbance of the excitability of the motor-nerves, and with well-marked elevation, or, at least, without any material diminution of the galvanic excitability of the muscles, speaks in favor of *amyotrophic lateral sclerosis;* this is always accompanied with paralysis and atrophy, but reflex action may be preserved.

The middle form of the reaction of degeneration, but with a greater or less degree of diminution of galvanic excitability of the muscles, which is hard to demonstrate and can only be shown in single muscles and groups of muscles, speaks in favor of *progressive muscular atrophy* (and *bulbar paralysis*). There is then no complete paralysis, non-uniform atrophy, while reflex action may be retained. It is true that the later stages of the disease, such as are so often found in the little muscles of the hand, cannot easily be distinguished by electrical examination alone from similar changes caused by poliomyelitis anterior or amyotrophic lateral sclerosis (nor from peripheral paralysis, lead paralysis, etc.), but then the diagnosis can usually be made from the other manifestations, and from the development and course of the disease.

At all events, it seems worth the while to pay further attention to these relations, as they are not only of diagnostic importance, but promise to throw light upon certain difficult problems in the physiology of the spinal cord which cannot easily be approached in other ways. To be sure, these examinations demand great practice, patience and technical knowledge, and I cannot too urgently recommend the greatest care in conducting test examinations. I can guarantee the facts discovered and communicated by me, but they urgently require to be extended and confirmed in numerous new observations.

The *further course* of chronic poliomyelitis anterior, after it has been developed to the point above described, may shape itself in various ways.

In the majority of instances the disease now *remains stationary for some time.* The paralysis increases no more, the weakness or impossibility of movement remains about the same; but the muscular atrophy does still advance. Now, too, moderate paralytic contractures may be developed, chiefly caused by the mechanical approximation of the points of attachment of the muscles. No other new disturbances arise.

After a while—a couple of weeks or months—very *gradual improvement* sets in, generally first in the upper extremities;

single movements first return, others again grow more distinct and stronger, and thus the improvement gradually advances, while the galvanic excitability of the muscles sinks more and more, and slowly gives place to the normal conditions of excitability, the contractures disappearing again little by little.

But the improvement is slow; it is only after the lapse of months that the patients can again eat, write, and use their hands for all kinds of manipulations.

Then the lower extremities have their turn; first the movements of the hip-joint become freer and more powerful, then those of the knee, and not till quite late those of the foot and toes. The course of things with reference to electrical excitability and the contractures is precisely the same as in the upper extremities.

The question of how far this improvement goes varies somewhat in individual cases. Not infrequently it advances to *complete recovery*, so that mobility is quite restored, the nutrition of the muscles again becomes normal, and patients regain all their former capabilities. This may, however, require many months and even years.

Incomplete recovery, however, is far more frequent. A part of the muscles (especially those in the domain of the peroneal nerve) remain paralyzed and atrophic, and patients retain the defect thus induced for life.

In a minority of instances the malady advances further and further upwards; finally, disturbances of respiration set in, signs of asphyxia, various bulbar symptoms, paralysis in the domain of the facial nerve, of the tongue, of swallowing, etc., and these manifestations gradually lead to *death*. Sometimes this likewise follows, without any further complications, through simple exhaustion.

The *duration* of the disease is, accordingly, in most cases, comparatively long; at best it is a question of months, generally of years. The progressive cases terminate fatally in from one to four years.

In view of the small number of observations thus far at our command, I may be permitted to communicate a brief outline of the following case, of a rather subacute form, recently observed by me.

Mr. A. G., forty-two years of age, was always of robust constitution and health, and has exposed himself with impunity to many injurious influences (heat, draughts, travels, the free use of alcoholic drink). For a year past he has often complained of unusual weariness.

His illness began at the end of July, 1876, with *general weakness*, headache, slight dyspeptic symptoms, etc. The patient still went about, notwithstanding increasing weakness of the legs, until the 22d of August, when, his legs giving way, he fell down-stairs. From this time on he was bed-fast. The only subjective troubles complained of were some *shooting* and *piercing pains in the legs*, with transitory *formication*.

At the end of September similar *weakness in the hands* set in, so that the patient could neither write, feed himself, nor do anything else with his hands. He never had the sensation of a tight band about his body. *The functions of the bladder and rectum were entirely undisturbed.* His general condition of late had been quite good.

Condition on examination, the 6th of October.—There was a high degree of *paresis and partial paralysis of the lower extremities.* Movements of the ankle-joint and toes were quite impossible, of the knee- and hip-joint quite difficult.

Sitting up in bed was very difficult. *The upper extremities were also to a high degree paretic*, especially the hand and forearm. The grasp of his hand could scarcely be felt; the extensors were completely paralyzed. The power of the muscles of the upper arm was materially lowered, that of the muscles of the shoulder least so.

The mobility of the muscles of the neck and face, of mastication, deglutition, and of the eyes was completely intact.

The *sensibility of the skin* in the feet and legs was somewhat, although but very slightly, dulled; from the thigh upward, as well as in the upper extremities, it was entirely normal, excepting a subjective sensation of numbness in the tips of the fingers.

Reflex action—of skin and tendons—in the lower extremities was *completely abolished.*

The muscles, which were very lax and flabby, were already *considerably atrophied* in all four extremities. The bladder and rectum acted normally. No bed-sores. Appetite and digestion regular. Sleep good. Head free.

Faradic excitability in the paralyzed muscles and the nerves belonging to them was greatly lowered, in part entirely abolished.

The *galvanic excitability* of the nerves of the lower extremities was completely extinguished; in the muscles, on the contrary, it was *raised* and *changed in quality* (twitching, sluggish, AnSZ>KaSZ); so at least in the domain of the peroneal and tibial nerves. In the domain of the crural nerve, diminution of the galvanic excitability could already be demonstrated with a continuation of the qualitative change. In the upper extremities the well-marked reaction of degeneration existed only in the extensors of the forearm and in the interossei muscles.

The *mechanical excitability* of a portion of the atrophied muscles was distinctly heightened.

The *treatment* consisted of the use of dry cups along the spine every couple o days, Priessnitz's cold-water compresses to the back, and iodide of potassium.

His condition very soon began to *improve*. At the *beginning of November* thi was distinctly perceptible in all the extremities, even though but to a very sligh degree.

At the *beginning of December* the patient was able to sit in a chair, and to mov his arms and legs much more freely. In the upper extremities the domain of th radial nerve was still completely paralyzed; a slight contracture of the flexors c the hand and of the fingers had set in. Movements of the elbow- and shoulde joint were now perfectly free and strong.

In the lower extremities there was still entire paralysis of the foot and the toe Extension of the knee-joint feeble; lifting of the leg possible; a slight contractu of the flexors of the leg.

Sensibility and the state of the bladder were normal. The paralyzed muscl still showed the reaction of degeneration.

He was now ordered galvanic treatment to the back and extremities.

His condition now steadily improved. At the *beginning of February*, 1877, th mobility in the extensor region of the forearm was pretty well restored; the co tracture of the flexors had almost entirely disappeared; the hands had greatl gained in strength, the patient could again feed himself.

The mobility of the muscles of the thigh was materially improved; the muscl of the legs were still completely paralyzed. The atrophy was somewhat less.

Electrical examination still showed the full reaction of degeneration, althoug now with distinctly diminished galvanic excitability.

Middle of March, 1877.—The mobility of the upper extremities was now almo entirely normal; the first signs of movement in the joints of the feet now began t appear; the general condition very good.

Middle of April, 1877.—Continued improvement. Movement of the feet an toes is becoming more distinct and extensive, although but very gradually. Th patient is now able to stand somewhat, and, if well supported, to move a few step The upper extremities are now almost entirely normal.

Diagnosis.

The picture of the disease in subacute and chronic poliomyelitis anterior has so much that is characteristic about it, that i cases which are at all well-marked it can very easily be recognized. Especial significance is to be attached to the *tout ensembl* of the disease and the successive development and consecution o the symptoms. This disease can generally be easily distinguished from all similar and kindred affections, notwithstanding the great similarity that frequently exists between them, by the lassitude, weakness, paresis, and finally paralysis, occurring at first in the lower and then in the upper extremities, the muscles being lax, without tension or contracture; by the progressive atrophy with the reaction of degeneration; by the failure of reflex action; by the absence of any disturbances of sensibility and of the bladder, as well as of any tendency to the formation of bed-sores; by its slowly progressive course and its usually favorable termination.

The differential diagnosis between this and *acute* anterior poliomyelitis can only come in question when it refers to the sequels of the latter affection. The existence of the latter is determined, however, by the fact of its extremely acute beginning, which at once causes the highest degree of paralysis. The subacute form has a decidedly progressive course: it extends more or less rapidly upward, the paralysis increases for a time, the disease may at last terminate fatally, or it may run a similar progressive course towards recovery. In both respects there seems to me to be such an essential difference between the acute and chronic forms that it appears to me doubtful whether the two actually represent the same process of disease developed with different degrees of acuteness.

Progressive muscular atrophy has very often been confounded with chronic anterior poliomyelitis. Notwithstanding all the resemblance which the two forms of disease may bear to one another in certain stages, they may easily be distinguished by the following characteristics: in chronic poliomyelitis there is first paralysis and afterwards atrophy; in progressive muscular atrophy the paralysis becomes associated with atrophy which has already long existed; in the former there is atrophy in the bulk of the muscles, in the latter only partial atrophy of the same; in the former the well-marked reaction of degeneration can be demonstrated as well as the failure of reflex action; in the latter, if there is any reaction of degeneration it is only the middle form, and reflex action is retained; the former runs its course comparatively fast and generally favorably, the latter very slowly and always unfavorably.

The distinction between this disease and *amyotrophic lateral sclerosis* is still more easy. The latter, it is true, resembles chronic poliomyelitis in the paralysis and atrophy in bulk of the upper extremities, but in the lower extremities it shows paralysi without atrophy, with tension of the muscles and contractures with increase of the reflex action of tendons, etc. Perhaps examination by electricity may also contribute something toward the determination of this question, if the fact should be furthe confirmed that in amyotrophic lateral sclerosis only the middl form of the reaction of degeneration occurs.

With regard to the distinction between this and the slowe forms of *Paralysis ascendens acuta*, see the following section.

Chronic anterior poliomyelitis will be capable of being easil distinguished from all other forms of chronic spinal disease from transverse chronic myelitis, multiple sclerosis, tabes dorsalis, spastic spinal paralysis, etc., if proper regard is paid t the condition of sensibility, of the bladder, of the nutrition of th skin, of reflex action, of electrical excitability, etc., quite irrespective of the other peculiarities of these various forms o disease.

In the well-marked and more diffuse forms, therefore, th disease is easy to recognize. But it is a question which can onl be determined by further successful investigations, whether another series of more partial forms of paralysis and atrophy o the extremities do not belong in the same category. It alread seems probable that this is, to a certain degree, the case in so-called lead-paralysis.

But I would also raise the question with regard to quite similar conditions of disease which have arisen spontaneously, viz with regard to partial paralysis with atrophy and the reaction o degeneration without disturbances of sensibility in the extremities; with regard to a good many cases which are cited in paper on progressive muscular atrophy, but which absolutely have no been progressive; whether all these do not owe their origin to quite circumscribed chronic anterior poliomyelitis. Of cours this question can only be solved by accurate clinical observations, made with this object in view, and finally settled only b pathologico-anatomical investigation. I therefore recommend i as worthy of further attention.

Prognosis.

The prognosis of this disease is never to be treated lightly; life may very easily be endangered, which never seems to be the case in the acute form.

At the same time the prognosis is comparatively favorable, as in the majority of cases recovery follows, and at all events the improvement, as a rule, goes much farther than in the acute form. At the same time patients must make up their minds to a comparatively long duration of the disease, extending perhaps over several years.

The prognosis is bad only in that form of the disease which advances upward with comparative rapidity. Whenever difficulty of breathing and of swallowing, labored action of the tongue, etc., set in, a fatal termination is at least very probable.

The more partial forms are never dangerous to life, but may lead to permanent disappearance of the muscles and corresponding defects of motion.

Therapeutics.

In view of the small number of observations as yet made, there is, of course, no question of any established, independent treatment of the disease.

In general, those principles are applicable which we have laid down elsewhere for the treatment of subacute and chronic myelitis generally. Therefore, at the beginning of the disease, we may employ abstractions of blood, derivatives of all sorts, hydropathic applications to the back, iodide of potassium, ergot, etc.

Undoubtedly at a later period the most that can be expected is from the galvanic current. It must be left to the future to determine whether mineral baths, cold-water treatment, or other similar means are of any special avail, and what internal remedies promise the best results. The best we can do at present is to continue our trial of the curative measures appropriate in chronic myelitis.

17. *Paralysis Ascendens Acuta.—Acute Ascending Paralysis. —Landry's Paralysis.*

O. Landry, Note sur la paralysie ascendante aiguë. Gaz. hebdom. 1859. Nr. 30 31.—*Kussmaul*, Zwei Fälle von tödtl. Paraplegie ohne nachweisb. Ursache Erlangen, 1859.—*T. L. Walford*, Softening of the Spinal Cord. Assoc. Med Journ. 1854. Nov. 11. (Canstatt's Jahresb. III. S. 83.)—*Gomes de Valle*, Ob d. paral. asc. aig. Union méd. 1861. Nr. 13.—*Leudet*, Paral. asc. aig., rapid mortelle, surven. dans l. conval. d. l. fièvre typhoïde. Gaz. des hôp. 1861. Nr 58.—*Pellegrino-Levi*, De l. paral. asc. aiguë. Arch. génér. 1865. I. p. 129.—*Bablon*, Obs. d. paral. asc. aig. Gaz. hebdom. 1864. Nr. 49.—*Gru*, Union méd 1866. Nr. 152.—*Caussin*, Gaz. des hôp. 1866. Nr. 23.—*H. Jones*, Brit. Med Journ. 1866. Oct. 27.—*Hayem*, Paralys. asc. aiguë. Gaz. des hôp. 1867. Nr 102.—*Harley* and *Lockhart Clarke*, Fatal Case of Acute Progress. Paralysis Lancet. 1868. Oct. 3.—*C. Lange*, Om obstigende spinalparalyse. Hosp. Tid 12. Aarg. Nr. 6-10. (Virchow-Hirsch, Jahresber. pro 1869. II. S. 34.—*Chevalet*, Par. asc. aig. d'origine syphil. Bullet. d. thérap. 1869. Oct. 15.—*Labadi Lagrave*, Gaz. des hôp. 1869. Nr. 148.—*O. Bayer*, Heilung einer acut. ascend Paral. unter antisyphil. Beh. Arch. d. Heilk. 1869. S. 105.—*A. Eulenburg* Lehrb. d. funct. Nervenkrankh. Berlin, 1871. S. 603.—*Reincke*, Fall v. Paral asc. acut. Deutsch. Klin. 1871. Nr. 23, 24.—*Bernhardt*, Beitr. z. Lehre. v. d acut. allg. Paral. Berl. klin. Woch. 1871. Nr. 47.—*Chalvet*, Gaz. des hôp 1871. Nr. 93.—*Leyden*, Klinik d. Rückenmarkskrankh. I. S. 94. II. S. 201 —*Herm. Levy*, Paralysis asc. ac. Correspondenzbl. d. ärztl. Ver. d. Rheinprov 1873. Sept. (Centralbl. 1874. Nr. 11.)—*Petitfils*, Considér. sur l'atroph. aig des cell. motrices. Paris, 1873.—*Eisenlohr*, Zur Lehre v. d. acut. spin. Paralyse. Arch. f. Psych. u. Nervenkrankh. V. S. 219. 1874.—*Calestri*, Gazz. Lombard. XXXIV. Nr. 20. 1874. (Schmidt's Jahrb. Bd. 168. S. 18.)—*Salomon* Schnelle Heilung einer schwer. ac. Rückenmarksaffection u. s. w. Correspondenzbl. d. ärztl. Ver. im Rheinl. 1875. Nr. 15.—*Goltdammer*, Ueber einige Fäll von subac. Spinalparalyse. Fall. 3. Berl. klin. Wochenschr. 1876. Nr. 26.—*Baumgarten*, Eigenthüml. Fall von Paral. asc. aiguë mit Pilzbildung im Blut Arch. d. Heilk. XVII. S. 245. 1876.—*C. Westphal*, Ueber einige Fälle vo acut. tödtl. Spinallähmung. Arch. f. Psych. u. Nervenkrankh. VI. S. 765. 1876 —*Déjerine* et *Goetz*, Paral. asc. aiguë. Arch. de Physiol. norm. et path. 1876 p. 312.—*R. v. d. Velden* (*Leyden*), Fall von acut. aufsteigend. spinaler Paralyse. Deutsch. Arch. f. klin. Med. XIX. S. 333. 1877.

History.

In the year 1859, Landry described some cases of disease under "Paralysie ascendante aiguë," which had terminated i death, with the manifestations of spinal paralysis advancing rapidly from below upward, and finally paralyzing the medulla oblongata, and in which no appreciable anatomical lesions were found. In the same year Kussmaul also described two cases observed by him of similar, rapidly fatal spinal paralysis, with entirely negative results on post-mortem examination.

No doubt cases of disease belonging to this category had previously been described in literature—thus, by Ollivier, in his work, under the head of Hyperæmia of the Spinal Cord, by Walford, and others. It appears that the distinguished Cuvier also died of this disease in 1832.

But it was not until after Landry's publication that reports of cases of this form of disease began to accumulate. Much, however, was included under this head which evidently does not belong here. Thus, cases of acute, central or diffuse myelitis (for instance, the cases of Levy, Picard, and others), or of subacute poliomyelitis anterior (cases of Behm and Taylor), or of infectious myelitis (Baumgarten); perhaps it is also wrong to place here those syphilitic affections which run their course presenting the picture of acute ascending paralysis.

We may learn how difficult it is to keep these different forms of disease separated when we remember that Petitfils has but recently made an attempt, in the most detailed manner, to establish the identity of acute ascending paralysis and acute or subacute anterior poliomyelitis,—to regard the former merely as a variety of the latter, and to refer it likewise to acute alterations in the large ganglion-cells of the gray anterior horns. In view of a series of well-established, more recent, and most recent facts, this theory may be regarded as overthrown.

Even before this theory was advanced, cases had repeatedly been observed which might be regarded as typical of acute ascending paralysis, and in which even the most careful examination by a skilled and practised hand discovered no trace of any change in the central nervous system (the cases of Vulpian, Pellegrino-Levi, Cornil and Ranvier, Hayem, Bernhardt, and others). The most recent observations of Westphal are decisive, and to a certain degree conclusive. In a number of cases that were clinically well marked, he could not find any trace of anatomical alterations in the spinal cord, even on the most careful anatom cal examination, carried out in every direction. Almost at th same time, Déjerine and Goetz published a case belonging her with the same negative anatomical result. Westphal has al rendered the clinical diagnosis of the disease more definite i various directions.

It is thus settled, for the present, that the disease cannot b considered as identical with any of the previously named form of spinal disease, but rather that it is a clinically well-chara terized—undoubtedly spinal—form of disease, the anatomica conditions of which are entirely negative, so far as our prese means of investigation show. The disease may, and must, ther fore, as yet be regarded as systemic; to be sure, we stand but the threshold of our knowledge thereof, and it will require add tional clinical, pathogenetic, and also anatomical investigatio to give us any satisfactory idea of the same. The material th far at our command is far too inadequate for this purpose.

Definition.

The disease designated by the name of *paralysis ascenden* *acuta* is clinically characterized by a motor paralysis (ascendin paralysis), which generally begins in the lower extremitie spreads pretty rapidly over the trunk to the upper extremitie and usually also involves the medulla oblongata, which som times runs its course without fever, sometimes with more or le active fever, which but slightly involves the general sensibilit and the functions of the bladder and rectum, and which runs it course without any notable atrophy of the muscles, and withou any diminution or change of their electrical excitability.

In the majority of instances the disease terminates fatally, b asphyxia, paralysis of deglutition, and the like; but lighte cases may also end in recovery.

The *anatomical* characteristics of the disease are at presen purely negative. No pathologico-anatomical alterations are t be found anywhere, and especially not in the spinal cord, whic might explain the picture of the disease. In particular an there no signs of hyperæmia within the spinal cord, of myelitis, of acute destruction of the ganglion-cells or nerve-fibres.

If the disease is therefore to be localized within the spinal cord at all, it is a question of finer, so-called impalpable disturbances of nutrition, not accessible to our present means of examination.

Etiology and Pathogenesis.

On this subject very little is known, and, in fact, nothing positive.

Sometimes the disease arises without any demonstrable predisposing or exciting causes.

Men are most frequently attacked ; among the sixteen cases collected by Pellegrino-Levi, only four were in women.

Most cases of the disease occur between the ages of twenty and forty, but it may also occur at later periods of life.

Nothing is known of any hereditary or neuropathic influences.

Taking cold is mentioned first in the line of exciting causes. It is not necessary here to mention the individual possibilities of the same, as they proved to be effectual in an entire series of cases.

Not a few instances have been seen to arise during the course of, or during the convalescence from, *acute diseases* (typhoid fever, diphtheria, pleurisy, varioloid, etc.). Some few have followed *suppressed menstruation* (through taking cold or emotional disturbances).

Bablow saw the disease break out after a man had indulged in the act of coition while standing up.

It is at least doubtful, as yet, whether *syphilis* is an actual cause of genuine acute ascending paralysis. We are accustomed always to find palpable alterations in syphilis, so that a syphilitic affection of the spinal cord with an impalpable lesion does not seem to us exactly probable ; at the same time, such a thing is possible. At all events, various authors (Kussmaul, Landry, O. Bayer, and others) have decided on the syphilitic nature of the disease, partly from the antecedents and partly from therapeutic results.

Westphal has recently taken up the idea of an intoxication as the cause of acute ascending paralysis. The same had already been announced by Landry, and briefly touched on by Hayem and Bernhardt. Baumgarten's case, in which the bacteria of splenic fever (Milzbrand) were found in the blood and in the spinal cord, appears to give some support to this supposition; still, in this case, there was undoubted myelitis present, therefore it probably does not belong here. Westphal considers it probable that some intoxication—thus far, to be sure, of an entirely unknown variety—is the actual and final cause of acute ascending paralysis. This can only be decided by numerous further observations.

It readily occurs to us to draw a parallel between this and another affection, likewise spinal, which sometimes terminates fatally within a few days, and sometimes runs a longer course to recovery, likewise appearing in lighter and severer attacks, and for which, up to the present time, there has likewise been found no anatomical lesion. We refer to *tetanus*. This parallel does not need to be carried out any further; but close observation cannot fail to detect the manifold analogies of the two forms of disease, although in the one (tetanus) it is a question principally of manifestations of motor irritation, and in the other (acute ascending paralysis) principally of manifestations of motor paralysis.

Pathological Anatomy.

The few convincing and sufficiently careful reports of post-mortems now before us (Vulpian, Cornil et Ranvier, Bernhardt, Westphal, Déjerine et Goetz) unanimously teach that there are absolutely no anatomical changes present to which the symptoms could, with any reason, be referred.

The examinations fulfil all the requirements that can be made of them at the present time; they have furnished *entirely negative results*, as well with reference to the brain as with reference to the spinal cord and the medulla oblongata, and likewise with reference to the sympathetic nerve, the peripheral nerve-trunks, and the muscles. The future alone can tell whether the changes in the anterior roots found by Déjerine and Goetz, but unfortunately not thoroughly enough examined, are of any significance. It is not probable that they are so.

On the other hand, the older investigations, which claimed to have proved the presence of hyperæmia here and there, of softening of the spinal cord, even of myelitis and the like, lose all their significance; at best they belonged to other clinically somewhat similar forms of disease. This also holds true of the very newest observation recently published by Leyden (v. d. Velden), under the name of acute ascending paralysis. This decidedly does not belong to the form of disease which we have here, in harmony with the views of Westphal and others, described under this name, as is abundantly evident from the clinical symptoms (a high grade of fever, delirium, paralysis of the bladder, loss of electrical excitability, stiffness of the back, tension of the muscles, etc.). What is here presented seems rather to be an admirable instance of acute disseminated myelitis, especially affecting the antero-lateral columns and the anterior gray substance.

It may be a matter of some significance that in single cases similar alterations have been found in the spleen, the liver, the lymphatic glands, and intestinal follicles, etc., as they are also so frequently found in diseases of infection—such changes as cloudiness, swelling, enlargement, more abundant blood-contents, etc.

Pathology of Acute Ascending Paralysis.

Symptoms.

In most instances, though not always, the appearance of the characteristic manifestations of paralysis are preceded by various *prodroma*, such as slight febrile action, general discomfort, pulling and shooting pains in the back and the limbs, paræsthesia of various kinds, as formication, numbness in the feet and finger-tips, and the like, generally also a feeling of great weariness and striking weakness. These may last for a longer or shorter period, for one or for several days, even a week and longer; such premonitory symptoms have been observed for as long as six weeks.

The actual *beginning of the disease* is marked by a striki *weakness of the lower extremities*, which increases more or le rapidly, either gradually or with sudden greater steps, and ve soon amounts to well-marked *paresis*. Standing and walki are soon rendered impossible, while when lying down the ind vidual movements of the legs can still be executed, although wi a growing diminution of force. Gradually these movements al fail, first in the joints of the feet, then in the knees, and finally the hips; and so in the course of a few days, seldom earlier later, there is *complete paralysis* of the limbs.

The legs then lie there, lax and immovable, show no traces muscular tension or contractures, exhibit no fibrillar twitching nor any other spasmodic manifestations, do not present th slightest resistance to passive movements, and are not subject pain, or but very slightly so, either spontaneously or on tou or pressure.

The paralysis now gradually advances upwards, uniform and steadily, but seldom interrupted by a prolonged arrest of th malady.

The *muscles of the trunk* are next attacked; sitting up an sitting straight are gradually rendered impossible by the paraly sis of the muscles moving the spinal column; the acts of expir tion, of coughing and sneezing, of defecation, etc., become wea and powerless through paralysis of the abdominal muscles.

Not long after—though here, it is true, the rapidity of th progress is very variable in different cases—the *upper extremiti* are also attacked with the weakness and paralysis. First th hands grow weaker and less steady, the strength of grip dimin ishes rapidly, ordinary acts, such as writing and feeding one self, grow impossible. Then the movements of the arms gro increasingly more difficult, finally the movements in the shoulde joint, too, become more or less feeble and paralyzed, so that a last the arms, like the legs, are entirely relaxed and immovable.

Then, too, *disturbances of inspiration* also appear, owing t paralysis of the intercostal and other respiratory muscles of th trunk.

When the disease has become developed up to this point, a objective examination shows very little that is marked, asid from the motor disturbances. As a rule, the very *slight disturbance of sensibility* is especially noticeable.

Subjective disturbances of the same, it is true, are not rare; patients complain of a feeling of numbness and formication in the fingers and toes, a diminution of feeling in the soles of the feet, rarely of higher grades, of loss of feeling in the lower extremities. There is generally no pain, or but an insignificant amount only at the beginning. The spinal column is not painful, the spinous processes not sensitive on pressure.

Objective tests of sensibility, in the majority of cases, give quite normal results; occasionally it is found slightly though distinctly lowered, especially towards the periphery of the extremities. More rarely higher grades of anæsthesia are demonstrated, and it may be a matter of doubt whether these cases belong in this category at all. These statements hold equally good for sensation of the skin and muscles.

In single cases a tolerably high grade of hyperæsthesia of the skin (hyperalgesia) has also been demonstrated.

At all events, however, the disturbances of sensibility play a subordinate rôle, in the picture of the disease, as compared to the motor paralysis.

No *ataxy* can be demonstrated, as long as any movements are capable of being carried out, although the feeble, trembling movements of the paretic limbs sometimes remotely remind us of this condition.

There is likewise no considerable *atrophy of the paralyzed muscles*. During the progress of the disease more or less considerable emaciation may appear, as in any one long confined to a sick-bed; but there is no question of a high grade of rapidly progressive atrophy, as in poliomyelitis anterior, and the few cases in which such has been reported were probably wrongly classified.

Another fact which stands in most intimate relation to the one just stated, and which appears to be of no less value in a differential diagnosis, is that *the electrical excitability of the paralyzed nerves and muscles remains entirely normal*. In all the more recent cases, carefully investigated by skilled hands (Pellegrino-Levi, Bernhardt, Westphal, etc.) no anomaly of electrical excitability worth mentioning has been found, even after th disease had existed for a number of weeks. By this very mean the disease seems to be distinguished, in a very significan manner, from all progressive paralyses caused by gross anatomical lesions within the spinal cord (myelitis centralis, poliomyeliti anterior subacuta, etc.). At all events the careful testing o electrical excitability is worthy of special attention in all futur cases.

But little is said with regard to the existence of *vaso-moto disturbances.* In Eisenlohr's case there was transitory œdem of the skin, with redness of the integument over various joints In some cases a profuse secretion of sweat is reported.

The nutrition of the skin does not usually suffer. Bed-sore do not occur.

The *conditions of reflex action* certainly deserve more carefu attention, as they are probably also of diagnostic significance Statements with regard to the reflex action of the skin are ver various; it also appears that this changes gradually during th course of the disease. It is but rarely that an elevation of refle action is reported (Eisenlohr's case); histories of cases generall show that reflex action is preserved during the first days and weeks of the disease, that it then diminishes more or less rapidly, and is finally entirely extinguished. If, as a general rule, reflex action thus entirely fails in well-marked and complete paralysis, still this does not seem to occur as rapidly and completely as it does, for instance, in poliomyelitis anterior. Special attention might be paid to this point. The reflex excitability of tendons has, thus far, only been examined by Westphal, who, in one case, found it, as well as the excitability of the skin, to be lacking.

The *condition of the sphincters* is a matter of no less importance. The bladder and rectum are generally quite undisturbed in their functions; there are no involuntary evacuations, there is no retention of urine—this is the rule. In individual instances, however, slight disturbances of the bladder occur; generally, however, even if they become more severe, they are merely transitory. Usually there is no trace of the severe paralysis of the bladder and rectum which occurs as a rule, for example, in central myelitis, and it is only exceptionally that the use of the catheter is required (cases of Déjerine and Goetz). In one case Pellegrino-Levi found the urine alkaline. There is usually constipation, which may, under some circumstances, become very obstinate.

Statements vary considerably with regard to the *general condition* of the patients; it may be more or less disturbed, although, in some instances, it is remarkably good. In the majority of instances there is said to have been no fever; at all events, it does not belong to the essential manifestations of the disease. In some cases active fever has been found, in others only a moderate degree of the same. At other times, again, single severe attacks of fever, with a chill, have been encountered; or there might merely be slight, transitory, febrile movement, extending over several days. This question, too, requires further more careful investigation.

In most striking contrast to the severe manifestations of spinal paralysis (for we can hardly regard it as being anything else) is the *complete integrity of the functions of the brain*, at first and during the earlier period of the disease. Intelligence and memory, consciousness and the functions of the special senses remain entirely normal; there never is any dizziness, delirium, and the like, and even complaints of headache belong to the rarities in this disease. Even the cerebral motor nerves either take no part at all in the general paralysis, or are not involved until very late. Actual paralysis of the muscles of the eye has not been observed; transitory amblyopia has been reported in two cases. Only those motor nerves which are directly dependent on the medulla oblongata are, as a rule, sooner or later involved in the paralysis.

After the disease has once reached the stage heretofore described, its *further course* may vary somewhat.

In most instances *the disease advances still further upwards.* The movements of the head and neck now begin to be disturbed; the muscles of the neck grow paralyzed; patients complain of weakness or of a feeling of stiffness in the neck; sometimes distinct weakness in the domain of the facial nerve sets in. Respiration grows increasingly difficult; a high degree of dyspnœa occurs, with very frequent and anxious respiration ; the activi of the diaphragm has been impaired, and the danger of asphyx draws threateningly nearer. Simultaneously herewith, or it m be earlier or somewhat later, disturbances of articulation of t voice, of speech set in, caused by paralysis of the muscular a paratus pertaining to these functions. To these are added wea ness and paralysis of the muscles of mastication, paresis of t soft palate (nasal speech, the regurgitation of fluids through t nose), difficulty of swallowing, and finally complete paralysis deglutition ; in a word, *the complete picture of severe bulbar pa alysis.* If we add that not infrequently differences in the tv pupils may be observed, and that the frequency of the pul often rises to a very alarming height, we shall therewith ha completed the clinical picture which indicates an advance of t process to the uppermost divisions of the cervical spinal cord.

All these manifestations continue to increase, while evidenc of hyperæmia and hypostatic congestion of the lungs app and the danger of asphyxia is augmented, until the *fatal term nation* is more or less rapidly ushered in.

The period of time which elapses before this termination reached may vary considerably. Sometimes it is but a fe days; the disease has been seen to run its course and end death within two or three days, although it may last for tw three, and four weeks; it seems to be rare for the disease extend over as long a time as six weeks. The average durati of fatal cases seems to be eight to twelve days.

But another termination is possible, having occurred in series of cases which may probably be counted as belonging this category, viz., *a termination in improvement and recover* Landry even speaks of eight recoveries in ten cases, and Pel grino-Levi found at least five cured out of fourteen diseased.

The disease may come to a stand-still at any stage of its d velopment ; then, some day, the paralysis extends no furth This generally appears to be the case before the paralysis h reached the nerves of the bulb, though instances have also be known in which recovery took place in spite of the existence disturbances of respiration, deglutition, and mastication.

The favorable turn usually takes place after the disease h lasted but a comparatively short time; some single movements appear to be a little better and stronger, others return once more; those parts last attacked by the paralysis are the first to show improvement. This gradually increases; the strength and power of function of the muscles grow from day to day; first the patients can use their hands again, gradually they learn to sit up, finally again to stand and walk. This may occupy a considerable time; it is always to be counted by weeks, and whether it be many weeks or few, varies very much in individual cases, and depends on circumstances entirely unknown to us. Usually the patients feel very weak and poorly for quite a while; fluctuations and relapses in this favorable course also seem to be possible.

Exceptionally the disease may be seen to *progress downwards* within the cord, instead of pursuing the more frequent ascending course. The paralysis then begins in the nerves of the bulb, and successively seizes first the upper and then the lower extremities. So in the case of the distinguished Cuvier, as reported by Pellegrino-Levi.

It may seem doubtful whether Westphal's Case 4 belongs under this head. This was likewise an instance of acute, fatal paralysis, with a perfectly negative result of post-mortem examination, but confined to the nerves of the bulb; there was paralysis of the tongue and lips, of deglutition, mastication, and respiration. This case would have to be considered as one of a more rudimentary character, more limited in its localization, and rendered rapidly fatal by this very localization. But it is probably better for the present to regard it as doubtful, and to leave it unclassified.

Diagnosis.

The picture delineated in the preceding pages is probably sufficiently characteristic to admit of a diagnosis of the disease being made in a fair proportion of the cases.

The paralysis, chiefly of a motor character, progressing rapidly from below upwards, the small amount of fever, the insignificant disturbances of sensibility, the absence of paralysis of the bladder, the slow extinction of reflex excitability, the absence of muscular tension and muscular atrophy, the non-occurrence of bed-sores, and the final involvement of the functions of the bulb would seem to be sufficiently indicative of this disea to point suspicion that way.

At the same time a positive decision will often not be p sible, especially during the first few days, and the diagnosis m for a time be unsettled. The affections with which acute ascen ing paralysis may be confounded are particularly the differe forms of acute and subacute myelitis, but on careful conside tion of all the symptoms one will generally be able—especial in the course of several days—to arrive at a tolerably certa decision.

This affection is hardly liable to be confounded with *acu poliomyelitis anterior* (section 15) because the latter has progressive character, never attacks the medulla oblongata, a never directly induces death; because it generally sets in wi fever, with very rapid loss of faradic excitability and rapid mu cular atrophy, etc. It is only in the light and temporary for of acute poliomyelitis anterior—which, however, are very rare that a doubt might perhaps arise. But these may perhaps distinguished from the light forms of acute ascending paraly by the entire failure of reflex excitability (?), and certainly m be distinguished by the lowering of faradic excitability—ev though it be but slight,—and by the non-progressive character the paralysis.

It is an easier matter to confound this malady with tho cases of *subacute* anterior poliomyelitis, which pursue a co paratively rapid course in an upward direction. Indeed th disease has, by a number of writers, been regarded as identic with Landry's paralysis. This is certainly incorrect, as w appear on a careful comparison of the symptoms of the two for of disease. The first argument in favor of the existence of acu ascending paralysis is to be found in the absence of atrophy a in the fact that the electrical excitability remains intact; th the longer preservation of reflex action, the possible presence slight disturbances of sensibility and of the bladder, the mo rapid and generally fatal termination, and the early appeara of severe symptoms of bulbar trouble, also point in the sa direction. Generally, however, we shall be able to conclu with certainty that a given case of paralysis is due to anatomi changes in the spinal cord, only on the development of atrophy and of the reaction of degeneration.

As a rule, Landry's paralysis can easily be distinguished from *acute central myelitis* (see above, p. 416). In the latter there is always a high degree of disturbance of sensibility, the early failure of all reflex actions, great paralysis of the sphincters, fever, acute bed-sores, a lowering of faradic excitability, and a rapidly fatal termination. Hence the distinction is not difficult.

The *spinal forms of syphilis*, which run their course under the form of acute ascending paralysis, may be distinguished from the latter, if, indeed, they are not identical with it, only by the evidence of former or still existing syphilis in the patient, and by the results of anti-syphilitic treatment.

Infectious myelitis, of which Baumgarten has certainly not given us a very complete clinical picture, ought to be distinguished from Landry's paralysis by the same signs which characterize any other diffuse or central myelitis.

The rare cases of *acute multiple neuritis*, one of which Eichhorst[1] has recently described, could only be mistaken for acute ascending paralysis by a very careless observer. The severe pains, limited to single nerve-roots, the anæsthesia and paralysis, similarly limited, and especially the rapid lowering of electrical excitability, should be enough to guard against this error.

Prognosis.

Acute ascending paralysis is always a disease of very doubtful prognosis. If it is far enough developed to be diagnosticated with certainty, then the prognosis is generally very serious. It becomes worse the more rapid has been the ascending course of the disease, the earlier respiration has been attacked, and the more pronounced the evidences of bulbar paralysis.

But we have seen that there are also exceptions to this rule, that no small number of cases even terminate favorably. It is difficult, however, to recognize these favorable cases early; as a rule, this is impossible until improvement has begun. Perhaps further experience will give us some fixed points for such a di tinction.

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[1] Neuritis acuta progressiva. Virchow's Arch. Vol. 69. 1877.

Therapeutics.

It is difficult, in view of our entire ignorance with regard the true essence of the disease, to decide on any definite ther peutic course.

One will be inclined, in general, to employ the treatment a plicable to the acute ascending forms of myelitis. But it is question whether the good results of this treatment have n been observed in those very cases which have been falsely dia nosed as acute ascending paralysis.

The main object of treatment certainly is to *bring about change in the nutrition of the nerve-elements of the spinal cor* This is an indication more easily set up than fulfilled.

It is more than doubtful whether the energetic application derivatives to the spinal column is justified by this indicatio The favorable results attained by Levy and Salomon with tl actual cautery occurred in cases which most probably did n belong under this head. At the same time, in so serious a di ease, one would not shrink from the frequent application of dr or wet cups, from repeated blistering, and, in case life wa threatened, even from the use of the actual cautery.

Careful stimulation of the activity of the skin is more likel to prove of benefit. This may be accomplished by frictions the skin with cold water, the repeated application of Priessnitz' "packs" to the trunk or to the entire body, lukewarm bath cold affusions, etc.

Of internal remedies, iodide of potassium is generally used a the nearest refuge in this therapeutic extremity, or nux vomica Their effects are doubtful.

An agent which promises much, and is certainly worthy o being more carefully tested, is the galvanic current. While it value has been established beyond a doubt for the later stage o the cases that terminate favorably, the question still remains t be decided, *when* its application is to be commenced. *A priori,* we would seem to be justified in beginning it very early, as ther is no fever, no inflammation, but merely an impalpable disturbance of nutrition in the spinal cord. And it is against just such a condition that the galvanic current, perhaps, shows itself especially efficacious. The application of a constant, moderately strong current the length of the spinal column might be tried first.

As a matter of course, these patients require the most careful nursing; their nourishment must be tonic and abundant; and on the appearance of symptoms of paralysis which threaten life, the free and energetic use of all the various stimulants (alcohol, coffee, musk, camphor, ammonia, nux vomica, etc.) must not be omitted.

In those cases which terminate favorably, subsequent treatment with electricity, baths, cold water, mountain air, etc., is indicated.

18. *Tumors of the Spinal Cord.—Intramedullary Tumors.*

Lebert, Traité d'anatom. patholog. Tom. II.—*Ollivier*, l. c. 3. éd. Tom. II. p. 503. 1837.—*Foerster*, Handb. der pathol. Anat. II. 2. Aufl. S. 628. 1863.—*Virchow*, Die krankhaften Geschwülste. Bd. 1 u. 2.—*Leyden*, Klinik der Rückenmarkskrankh. I. S. 468. 1874.—*M. Rosenthal*, Klinik der Nervenkrankh. 2. Aufl. S. 349. 1875.—*Hasse*, l. c. 2. Aufl. S. 729–735. 1869.

Eager, Arch. génér. 1834. Tom. 4. p. 605.—*Brown-Séquard*, Course of Lectures, etc. Phila. 1860. p. 60, p. 101, etc. Paralysis of the Lower Extremities. London, 1861. p. 92–99.—*Gendrin*, Tubercule comprimant, etc. cité par Brown-Séquard. Journ. d. l. Physiol. T. VI. p. 233.—*W. Gull*, Cases of Paraplegia. Guy's Hosp. Rep. VIII. S. 113. 1858.—*Schueppel*, Gliom u. Gliomyxom des R.-M. Arch. d. Heilk. VIII. S. 113. 1867.—*W. Scholz*, Paraplegie u. s. w., Bindegewebsneubildung im R.-M. Allg. militärärztl. Zeitung, 1868. Nr. 28—*John Grimm*, Ein Fall von progress. Muskelatrophie. Virch. Arch. Bd. 48. S. 445. 1869.—*E. K. Hoffmann*, Gliomyxom im oberen Theil des Lendenrückenmarks. Zeitschr. f. rat. Medic. III. Reihe. Bd. XXXIV. S. 188. 1869.—*J. Sander*, Fall von Paralysis atrophica. Arch. f. Psych. u. Nervenkrankh. II. S. 780. 1870.—*Habershon*, Clinical Cases, etc. Tubercle in the Spinal Cord. Guy's Hosp. Rep. XVII. p. 428. 1872.—*G. Hayem*, Tubercule d. l. moelle ép. Arch. d. Physiol. V. p. 431. 1873.—*Charcot*, Leçons sur les maladies du syst. nerveux. II. Sér. 2. Fasc. 1873.—*Chvostek*, Zwei Fälle von Tuberkulose des R.-M. Wien. med. Presse, 1873. Nr. 35. 37–39.—*Westphal*, Fall von Höhlen- und Geschwulstbildung im R.-M. Arch. f. Psych. u. Nervenkrankh. V. S. 90. 1874. —*Th. Simon*, Ueber Syringomyelie u. Geschwulstbildung im R.-M. Ibid. V.

S. 120. 1874.—*Liouville*, Nouv. exempl. de lés. tubercul. dans la m. ép. Ar génér. 1875. Janv. p. 92.—*Klebs*, Beitr. z. Geschwulstlehre. Leipzig, 1877. 89. Prag. Vierteljahrschr. Bd. 126 u. 138.

In this section we shall consider those pathological neoplas which are developed within the substance of the spinal co itself, in so far as they occur in the form of tumors, and are me or less sharply distinguished from the substance of the cord.

Tumors of the substance of the spinal cord are very ra affections; they are also, clinically, very difficult or impossib to recognize with certainty, and therefore seem to be of but litt practical importance; consequently, we shall treat the subje briefly.

Pathological Anatomy.

There are but few forms of neoplasm that have thus far bee observed in the spinal cord itself, and but single ones of the have acquired some degree of practical importance through t comparative frequency of their occurrence. Such are glioma and tubercle of the cord.

The majority of medullary neoplasms appear to be develope in the midst of the substance of the spinal cord; only in rar instances do they grow from the spinal meninges into the cor It cannot as yet be determined whether the white or gray matt of the cord affords the principal soil for the development of th various neoplasms. Some writers assert one thing, some anothe Perhaps the relations vary according to the various forms o neoplasm. According to the observations of Th. Simon, it woul almost appear as though the anterior portions of the white pos terior columns were the favorite seat of some neoplasms. Thi much, however, is certain, that intramedullary neoplasms ma be developed from the most varied points in the gray and whit substance of the cord.

The size of the tumors can never be very considerable, on account of the narrow space within the spinal canal. They are generally round or longish growths, from the size of a hemp-seed to that of a hazel-nut, or even of a thumb. In single cases, however, extraordinarily elongated tumors have been found, which have stretched, in various degrees of thickness, throughout the entire cord, from the conus medullaris to the medulla oblongata.

The substance of the spinal cord is at first only crowded apart by the tumors, more or less compressed, and finally caused to disappear entirely; more rarely the substance of the cord passes directly into the mass of the tumor. An inflammatory softening is always developed in the neighborhood of the neoplasm, a transverse myelitis, whence in many instances the first severe clinical symptoms proceed. Aside from this, the secondary ascending and descending degenerations so often spoken of are generally developed.

In the tumors themselves processes of softening are not rare; the same is true of the secondary development of cavities (syringomyelia), extravasations of blood, etc., as we shall see when studying the individual forms of the affection.

Among the tumors thus far observed in the cord, glioma is probably the most frequent variety (cases of Sander, Simon, Case VII.; Schueppel, Case I.; Klebs and others). It usually appears as a soft, grayish-red or grayish-white mass, of a roundish or more elongated shape, often very long drawn out and irregular in form. In many places it is merged impercebtibly in the surrounding tissue. It is very vascular, disseminated with numerous larger and smaller blood-vessels, and not rarely shows more or less extensive hemorrhages into its substance and in its neighborhood. The secondary development of cavities is very frequent in gliomata, especially of the softer form, and part of the cases of disease described under the name of syringomyelia evidently belong here (Westphal, Th. Simon). The very vascular forms have probably also been described as *teleangiectatic gliomata.* This variety, in particular, shows especially extensive hemorrhages.

According to Virchow, gliomata proceed from the neuroglia, and represent nothing else than a hyperplasia of the neuroglia. They consist of cells which are imbedded in a granular or fibrillated, or sometimes also in a more mucous basement membrane. Their wealth of cells varies more or less; their wealth of vessels is generally very considerable. The nerve-elements (fibres and cells) have generally entirely disappeared within the tumor, according to the reports of the majority of authors (Vircho Charcot, and others).

On the other hand, Klebs has recently undertaken to show that all these tum principally proceed from the nerve-elements, partly from true ganglion-cells, pa from medullated nerve-fibres, and that associated herewith there is a mighty de opment of blood-vessels and lymph-vessels. He therefore calls these tumors *neu gliomata*. It is claimed that the interruption of nervous conduction occurs m later in these hyperplastic tumors than in those forms of tumor which act u the nerve-tracts through mechanical pressure. This might be of value from a c ical point of view. Further investigations may reconcile these differences.

Gliomata of the spinal cord are said to occur principally the cervical portion of the same, and, according to Schueppel, arise chiefly from the gray substance.

Myxoglioma (Simon, Case VIII.; Schueppel, Case II.; Ho mann) is to be regarded as a variety of glioma. It is a brig red, translucent, viscid tumor, in which the cell-elements are i bedded in a basement substance containing mucine, and wh otherwise acts just like the simple glioma, with which the my glioma is sometimes combined. The same is true of *gliosarcom* of which Westphal has described one case.

Pure *sarcoma* (Foerster, Tuengel in his clinical communi tions, Hamburg, 1864, p. 27) is certainly a very rare form of tum within the spinal cord. Virchow has never seen an instance of *Myxosarcoma* has been observed here and there as a long-dra tumor with the development of a number of central cavit The cases of Hutin and Scholz, and the case of Sonnenkalb, ported in Ollivier, l. c., II., p. 402, perhaps belong in the ca gory of *fibrosarcomata*.

On the other hand, *tubercle* is one of the most frequent tumo perhaps the most frequent, of the spinal cord (Charcot). It h been observed in numerous cases (Gull, Eisenschitz, Vircho Hayem, Liouville, Habershon, Chvostek, and others).

Tubercle of the spinal cord occurs at all periods of life, m frequently between the ages of fifteen and forty, and compa tively oftener during youth.

These growths may be developed in all portions of the co but are especially fond of the two enlargements of the same, a according to Hayem are especially liable to occur in the lum enlargement. Sometimes they occupy principally the white, and sometimes principally the gray substance. Their size varies from that of a hemp-seed to that of a hazel-nut, or larger; they generally occur singly, rarely several are found at once, or a whole group of them. Tubercle of the spinal cord is seldom the only localization of tuberculosis within the body; there is almost always simultaneous tubercle of the brain, or tuberculosis of the lungs and other organs of the body can be demonstrated. Very generally gray miliary tubercles are at the same time to be found in the meninges of the cord.

Tubercles of the spinal cord have quite the appearance of the well-known, far more frequent tubercles of the brain; they are roundish, hard tumors, of a dry quality, of yellowish-white, cheesy color, of a lamellar arrangement on their cut surface; the peripheral layers are often more gray and hard, and contain young tubercle granulations. Sometimes a central cavity is met with, containing softened matter of a cheesy or pasty consistence (Chvostek, Habershon).

In the vicinity of the neoplasm there is always more or less extensive softening of the spinal cord, of an inflammatory nature; a secondary myelitis which is especially pronounced in the gray substance, and extends to a variable degree upward and downward within this substance. Sometimes, too, a species of encapsulation through sclerotic tissue is met with in the vicinity. According to circumstances—according to the position of the tumor—there will be secondary ascending or descending degeneration.

Syphilomata, gummata, are, on the whole, but rarely found within the spinal cord (cases of E. Wagner, Moxon, Charcot and Gombault, and others). They are then generally multiple, and occur simultaneously also in the brain. They have the same peculiarities as other gummata of the central nervous system.

Carcinoma seems never to occur primarily in the spinal cord, if we ignore the earlier, very doubtful reports with regard to "cancerous" new growths in the cord. Even the case of tumor of the cord, reported by John Grimm as medullary carcinoma, is probably somewhat questionable. Very frequently, however, carcinomatous growths, taking their origin from the vertebræ, grow into the spinal cord, but then they run their course qui accord with the clinical picture of meningeal tumors.

Probably those alterations within the cord which have described under the names of hydromyelus and syringomy may also be in part counted among tumors of the cord. Altho they are not exactly rare, they are of very little practical im tance on account of frequently running their course entirely u companied by any symptoms. Their method of developn seems to be various; sometimes there is an actual malformat sometimes a secondary widening of the central canal, someti a secondary development of cavities, such as occur from proce of softening in myelitis, or more frequently (Th. Simon) in ac neoplasms (gliomata, myxogliomata, myxosarcomata, etc.).

Etiology.

The causes of intramedullary tumors are as yet quite obsc Naturally enough, here too, as in meningeal tumors, the bl has been attached to all sorts of conditions, but it is gener very hard to tell with what degree of justice.

Traumatic lesions, a blow, a jar, a fall upon the spinal umn, are among the causes, the efficacy of which we should most inclined to recognize.

The influence of *pregnancy* and the *puerperal state* are q uncertain. These might more readily be believed to give occa for the appearance of the first symptoms where the neopl already existed but was as yet latent.

It cannot be regarded as settled whether severe *fright*, longed *trouble*, and the like, are capable of causing the devel ment of a tumor of the spinal cord.

It is certain, however, that some general diseases and dys sias are capable of occasionally becoming localized in the spi cord. This is true of scrofula and tuberculosis, as shown tubercle of the cord; of syphilis, as shown by spinal gummat

We know of nothing further to say with reference to the et ogy of intramedullary tumors.

Symptomatology.

The picture of disease by which tumors of the spinal cord betray themselves is exceedingly variable in different cases; it is manifold, and generally at the same time so uncertain that there can ordinarily be no such thing as a positive recognition of the same. This lies in the very nature of the case.

Either a more or less complete paraplegia appears, with all the usual signs observed in every myelitis from compression (paralysis of motion, sensation, and of the bladder, increased reflex action; finally, atrophy, bed-sores, etc.). The development of this paraplegia sometimes, though by no means always, occurs with lively excentric pain, shooting pains in the limbs, constricting pains like a girdle, etc., similar to the symptoms in meningeal tumors, with paræsthesia, local atrophy, and the like; sometimes it comes on with vague and uncertain symptoms (paræsthesia, weariness, stiffness, slight atrophy, etc.) lasting for a long time, until the paraplegia then suddenly and somewhat rapidly develops itself, evidently caused by a rapidly spreading transverse myelitis, or it may be by hemorrhage into the tumor itself and its surroundings.

Or a quite partial paralysis is at first developed, which may remain for a long time confined to one upper extremity, for instance, and then, in an irregular manner, seize the other extremities until the paraplegia has become complete. In such cases, too, the symptoms of a unilateral lesion have been observed for quite a long while (cases of Scholz, Gendrin), which have then, in the further progress of the affair, developed into complete paraplegia. Or various other groups of symptoms are called forth (progressive muscular atrophy, symptoms of tabes, signs of spasmodic spinal paralysis, etc.) according to the position, seat, and principal direction of development of the tumor.

In not a few cases the manifestations during life are exceedingly insignificant, and one is astonished, on making a post-mortem examination, to find such extensive changes in the spinal cord. This seems to be especially the case when a tumor quite centrally developed crowds the substance of the cord apart, and does not entirely destroy it. Th. Simon has even reported se eral exceedingly remarkable cases which ran their course entirel without any symptoms.

But when it is not a question of just such quite exception instances, then the picture of the disease is usually that of transverse myelitis, being more or less rapidly developed a extending over a transverse section of the cord. Sometimes t disease runs a more acute, sometimes a more chronic form, and is but seldom that in this picture the presence of a tumor can recognized as the cause of the transverse myelitis.

In some single instances the disease also appears under t picture of a slowly ascending, chronic, spinal affection, whic runs its course under the most complicated manifestations, a does not present the picture of the previously described chron diseases of the general system; therefore, represents more t picture of a diffuse ascending myelitis. In still rarer instanc one may also recognize a descending extension of the neoplas or of the myelitis caused thereby, within the gray substance, the subsequent disappearance of reflex excitability, the occu rence of extensive muscular atrophy, etc.

The attempt has thus far been made in vain to secure, fro amongst the individual symptoms, at least a few fixed poin on which to base the diagnosis of intramedullary tumor The following have been claimed as such. A somewhat lon antecedent history of active local manifestations of irritatio belt-like pains, excentric pains, definite paræsthesias, local para yses, as in meningeal tumors; early and well-marked atroph which points to a larger involvement of the gray substance striking fluctuations in the course of the disease, spontaneou improvement, and equally spontaneous growing worse agai (Schueppel); and finally, Schueppel has also tried to connec the occurrence of scoliosis (curvature of the spinal column to th side on which the tumor is situated) with the presence of tumor. In this he is doubtless wrong, as this manifestatio merely depends on unilateral paralysis of the muscles of th back, which may depend on all sorts of causes. Whoever is a all familiar with the story of chronic spinal diseases will readil see that all these symptoms are of but very subordinate signifi cance, and will never justify a positive conclusion as to the presence of a tumor.

The *termination* of tumors of the spinal cord is doubtless in the majority of instances fatal, with the single exception of syphilomata, in which energetic specific treatment may bring about a cure.

The fatal termination may be brought about in various ways. After the disease has perhaps remained latent for a considerable time, or has only betrayed itself by slight and insignificant manifestations, an acute aggravation thereof suddenly occurs, and the disease then runs its course to death under the picture of an acute or subacute transverse myelitis.

Or the affection progresses slowly, more and more complicated and severe symptoms showing themselves gradually; the evidences of paralysis take an ascending course, disturbances of respiration, paralysis of deglutition, etc., are added thereto, and bring about the end.

Or death is induced by intercurrent diseases developed during the progress of the spinal difficulty (typhus, variola, and the like), to which the patient is perhaps able to offer less than the usual amount of resistance.

The *duration* of the disease is not easily determined, as we can generally arrive at no certainty with regard to the beginning of the development of the tumor, unless very positive causes for the same can be demonstrated. As a rule, however, but a few years elapse from the beginning of the symptoms to the fatal termination—sometimes but a few months.

Diagnosis.

It is evident, from what has been said above, that the diagnosis of an intramedullary tumor is but very rarely possible. As a rule, one will have to be content with making out a distinctly localized, more or less extended, chronic spinal affection, under which one may only in exceptional cases, with a certain degree of probability, conjecture the presence of a tumor. This probability may rest upon the etiological conditions which may happen to be present; upon the demonstration of some previous traumatic cause, or existing scrofula, or tuberculosis, or syp lis. Among the symptoms those which perhaps deserve spec consideration are the fluctuations in the intensity of the para tic manifestations, the not complete interruption of spinal c duction; furthermore, it may awaken suspicion of a tumor the evidences of central myelitis or of hæmatomyelia superve upon those of a long-continued, insidious spinal affection.

If once the general presence of a tumor within the spi canal has become probable, it will still be difficult to distingui an intramedullary from a meningeal tumor. An argument favor of the former will be found in the slighter intensity of initial signs of irritation and in the frequently occurring grad upward advance of the disease. A positive conclusion, howev will but rarely be possible.

Prognosis.

The prognosis of spinal tumors—with the exception of syphilomata—is, of course, absolutely unfavorable. In the jority of cases, however—in view of the defective diagnosis— is first clearly brought to our view by the fatal termination of case.

Therapeutics.

Consistently with the above statements we have to admit t our therapeutics are generally quite hopeless. It is only w syphilis exists that we shall attain any results by energetic m ication directed against this disease. We know at present of remedy for the other neoplasms. And yet we would not for t reason oppose any attempt that may be made with iodide potassium, arsenic, iodide of iron, cod-liver oil, etc.

As a rule, we shall have to confine ourselves to the treatm directed against subacute and chronic myelitis, which has b explicitly set forth in previous sections, until we have convin ourselves and the patient of the hopelessness of all further tempts at a cure.

19. *Secondary Degenerations of the Spinal Cord.*

L. Tuerck, Ueber ein bisher unbekanntes Verhalten des R.-M. bei Hemiplegien Zeitschr. d. Ges. d. Aerzte in Wien 1850. Heft. 1.—*L. Tuerck*, Ueber secundäre Erkrankung einzelner Rückenmarksstränge und ihrer Fortsetzungen zum Gehirn. Sitzungsber, d. kais. Akadem. d. Wiss. Mathem.-naturw. Classe. Bd. VI. S. 288–312. 1851 und Ibid. Bd. XI. S. 93. 1853.—*Leyden*, Ueber graue Degener. des R.-M. III. Deutsch. Klin. 1863. Nr. 13.—*Bouchard*, Des dégénerations secondaires d. l. moëlle épin. Arch. génér. de méd. 1866. Vol. I. pp. 272, 441, 561. Vol. II. p. 273.—*Charlt. Bastian*, Case of Concussion-lesion with Extensive Secondary Degeneration of the Spinal Cord, etc. Med. Chir. Trans. L. pp. 499. 537. 1867.—*H. O. Barth*, Ueber secund. Degener. des R.-M. Arch. d. Heilk. X. S. 433. 1869.—*Vulpian*, Expériences relat. à la pathogénie des atrophies second. d. l. m. ép. Arch. d. Physiol. II. p. 221. p. 661. 1869. III. p. 521. 1870.—*Westphal*, Ueber ein eigenth. Verhalten secundär. Degenerat. des R.-M. Arch. f. Psych. u. Nervenkrankh. II. S. 374. 1870.—Ueb. künstl. erzeugte secund. Degener. einzelner Rückemarksstränge. Virch. Arch. Bd. 48. S. 516. 1869. und Arch. f. Psych. u. Nerv. II. S. 415. 1870.—*W. Mueller*, Beiträge zur pathol. Anat. und Physiol. des R.-M. Leipzig. 1871. Beob. 1.—*C. Lange*, Forelaesninger over Rygmarv. Patologi. 1. Heft. Kopenhagen. 1871. Fall von Myelit. interstit. chronica. Hosp. Tid. 14. Aarg. 1871 (s. Virchow-Hirsch, Jahresber. pro 1871. Bd. II. S. 77).—*C. Lange*, Om lednings forholdene i Rygm. Bagstränge, etc. Nord. med. Arkiv IV. Nr. 11. 1872 (s. Virchow-Hirsch, Jahresber. pro 1872. Bd. II. S. 79).—*Th. Simon*, Tumor im Sack der Dura spin., die Cauda equina comprimirend, mit fortgeleit. Degener. d. Hinterstränge u. s. w. Arch. f. Psych. u. Nerv. V. S. 144. 1874.—*Leyden*, Klinik der Rückenmarkskrankh. II. S. 301–317. 1876.—*F. Schultze*, Zur Lehre von der secund. Degener. des R.-M. Centralbl. f. d. med. Wiss. 1876. Nr. 10. —*A. Pitres*, Atroph. muscul. consécut. à une sclérose descend. Progrès méd. 1876. Nr. 8.—Des dégénérat. second. d. l. m. épin. dans les cas de lésions corticales. Gaz. méd. de Par. 1877. Nr. 3.—*Charcot*, Leçons sur les localisat. dans les malad. du cerveau. 1. fasc. 1876. p. 145–168.—*P. Flechsig*, Die Leitungsbahnen im Gehirn und R.-M. des Menschen. Leipzig, 1876. S. 230 ff.—Ueber Systemerkrankungen im R.-M. Arch. d. Heilk. XVIII. S. 101 S. 289. 1877.—*P. Schiefferdecker*, Ueber Regeneration, Degenerat. u. Architectur des R.-M. Virch. Arch. Bd. 67. S. 542–614. 1876.

A. Vulpian, Influence de l'abolition des fonct. des nerfs sur la région d. l. m. épin., qui leur donne origine. Exam. d. l. moëlle dans des cas d'amputat. d'ancienne date. Arch. d. Physiol. I. p. 443. 1868.—Sur les modific. qui se produis. dans l. m. épin. sous l'influence de la section des nerfs d'un membre. Ibid. II. p. 675. 1869.—*Dickinson*, On the Changes in the Nervous System which Follow the Amputation of Limbs. Journ. of Anat. and Physiol. Nov. 1868.—*Friedreich*, Ueber progress. Muskelatrophie u. s. w. Berlin, 1873. S. 138.—*Ley-*

den, l. c. II. S. 314. 1876.—*A. Gensmer*, Veränderung im R.-M. eines Amp tirten. Virch. Arch. Bd. 66. S. 265. 1876.

G. Hayem, Des altérations d. l. moëlle, consécut. à l'arrachement du nerf sciatiq chez le lapin. Arch. d. Physiol. V. p. 504. 1873.—Compt. rend. Vol. 78. 291. 1871.

History.

We are indebted to L. Tuerck (1851 and 1853) for the disco ery and the most thorough examination of certain secondar processes of degeneration which frequently occur within the sp nal cord. He was the first to make extensive reports with regar to these processes, and his observations, which, in spite of th defective methods of examination then employed, still pass mu ter at the present day, gave us a well-nigh exhaustive explana tion of the state of things, to which the most recent researc has added but little.

Some few observations belonging under this head doubtle received passing notice even before Tuerck's time; thus, fo instance, in the "Sepulcretum" of Bonetus. Cruveilhier als found secondary atrophy extending to the pons and the pyr mids in diseases of the brain, though he found nothing of this i the spinal cord itself. Rokitansky had the same experienc Tuerck, however, was the first one who thoroughly and almo definitively cleared up the question of secondary degeneratio within the spinal cord. His statements, however, chiefly ref to the localization and extension of the process under vario circumstances.

The first somewhat more accurate teachings with regard the histological conditions in secondary degeneration are tho of Leyden, in 1863.

After various French and other observers (Charcot, Corni and others) had published single cases which belonged under th head, Bouchard (in 1866) issued a work treating of this questi in all its phases, in the most masterly manner, wherein he n only sets forth the pathologico-anatomical relations, but al most specifically the pathogenesis and symptomatology of se ondary degenerations.

The communications which followed, consisting chiefly of reports of cases, developed nothing new that was of any significance. The only thing of importance was the demonstration furnished by Westphal—previously sought in vain by Vulpian, but afterwards confirmed by him—that secondary degeneration could also be experimentally produced in dogs. This is a fact of which, alas! no one has as yet availed himself for the purpose of submitting to an experimental test various still obscure questions connected with secondary degeneration.

Quite recently again the doctrine of secondary degenerations has been materially advanced, and in some respects cleared up, by two important and larger works. Flechsig, in his comprehensive work on the paths of conduction within the human spinal cord, has also examined and tested secondary degenerations more carefully, has compared the results of this testing with the very noteworthy results of his investigations into the history of development, and has found them to agree in a way that is entirely satisfactory.[1] Schiefferdecker too, has, at least in one direction, most carefully worked up a large amount of experimental material (in dogs whose spinal cord von Goltz and Freusberg had divided at a certain point, for purposes of physiological investigation). He has fully confirmed the former doctrines and somewhat extended them.

In spite of all this, many a problem still remains to be solved, and although the examination of secondary degenerations has already been of the greatest value in advancing our knowledge of anatomical, physiological, and pathological problems, yet just at this very point there is still a rich field for experimental investigations, which will undoubtedly lead to numerous and important results.

Etiology and Pathogenesis.

Since the investigations of Tuerck, it is known that certain diseases of foci lying *outside* of the spinal cord (especially within the brain), and having a definite seat, are followed by a second disease of quite definite paths of conduction, which disease extended for great distances into the spinal cord ; and that si larly, in certain diseases *within* the cord, secondary disease quite definite paths of conduction extends upward as well downward, far beyond the limits of the original focus of disea Finally, the same thing, even though to a more limited degr holds true of certain diseases of the peripheral nerve-tracts.

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[1] In his work, in the *Archiv der Heilkunde*, which is as yet but partly made public, *Flechsig* returns, in a more detailed manner and with more positive declarations, to the question of secondary degenerations.

Therefore, in diseases which are either developed in its o substance or in some portion of the nervous system lying outs of the same, the spinal cord, in a portion of its tracts, is secon rily involved in disease, and, indeed, as has appeared on more ca ful observation, this occurs in a perfectly regular manner a according to certain laws. Those changes in the spinal c which are thus caused are designated as *secondary degene tions*. They act quite differently, according to the seat of lesion which causes them. We must therefore distinguish se ral groups.

a. Secondary degeneration of the spinal cord in diseases the brain. This is, of course, always *descending* in its progre and is as good as exclusively confined to the so-called "pyra idal tracts" (Flechsig); that is, on the one hand, to the mo conduction-tracts united into a large bundle in the posterior h of the lateral columns ("Pyramidenseitenstrangbahnen"); the other hand, to the uncrossed pyramidal tracts lying togeth usually in a narrow bundle at the inner surface of the anter columns ("Pyramiden-Vorderstrangband"—*Flechsig*, "Hüls Vorderstrangband"—*Tuerck*).

This degeneration can be followed upward through the py mids and the pons into the crura cerebri and the fibrillations the inner capsule; it extends downward, always growing n rower and becoming limited to a smaller number of fibres, i the lumbar portion of the cord, to disappear gradually in lower half of the same.

Descending secondary degeneration of the pyramidal tra occurs in all destructive diseases of the brain which involve direct motor paths between the pyramids and the corona radi (perhaps more accurately the direct motor tracts of the in capsule—Charcot). This degeneration is called forth by effusions of blood, foci of softening, neoplasms, scleroses, and chronic inflammations, which to any considerable extent involve the pons, the peduncles, the optic thalamus and corpus striatum, the lenticular nucleus, etc. It has also been found in chronic hydrocephalus (F. Schultze). An indispensable prerequisite to the occurrence of this degeneration, however, seems to be that the direct motor tracts which run from the pyramids to the inner capsule[1] must be involved in the lesion; affections which are rigidly confined to the gray substance of the lenticular nucleus, the optic thalamus or the corpus striatum, call forth no secondary degeneration (Charcot).

It is furthermore demonstrated that lesions of any considerable extent in the *centrum ovale*, in case they are not situated too far from the foot of the corona radiata, call forth this degeneration.

Finally, it appears, from both older and more recent observations (Tuerck, Bouchard, Charcot, Pitres, Flechsig), that this descending degeneration also takes place in diseases of the cortex of the brain (if they are not very superficial only); but this is only true when such lesions are situated in the so-called "motor sections" of the hemispheres—in the central convolutions and their immediate neighborhood. Here even a comparatively small focus of disease is sufficient to cause the degeneration, while in the *non*-motor portions of the brain-cortex even larger lesions exist *without* secondary degeneration.

In all these brain affections, therefore,—and they constitute no small number of brain diseases generally,—after the lesion has existed for some time, descending degeneration of the pyramidal tracts takes place, and in the spinal cord this is situated on the opposite side from the brain-lesion in the lateral columns

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[1] I see by the continuation of *Flechsig's* work in the *Archiv der Heilkunde*, which has appeared since the completion of my manuscript, that the statements of *Charcot* and *Flechsig* materially differ with regard to the position of the pyramidal tracts within the inner capsule. *Charcot* places them in the anterior, *Flechsig* in the posterior divisions of the inner capsule. This question, which can doubtless be definitely settled by further investigations, is of primary interest in connection with the pathology of the brain.

(therefore crossed) and on the same side with
the anterior columns. Flechsig has shown th
are doubtless physiologically of the same signi
and that the pyramidal tracts are distribut
lateral and anterior columns, so that in one
idal tracts run through the crossed lateral
case a great part of the same take their way t
column of the same side. In secondary dege
the circumstances of this distribution may als

The case made public by F. Schultze must, thus far, be
alone. In a sarcoma of the anterior division of the corpu
scending degeneration in the external posterior columns
culus cuneatus" of Burdach], reaching down to the dors
while Goll's columns and the lateral columns were free.
absence of the greater part of the axis-cylinders and marke
remained; the medullary sheath was chiefly preserved, the
no proliferation of nuclei, no granule-cells, no fatty dege
therefore a picture differing from the one usually found in
The entire condition of things in this case is unique, if
may therefore seem doubtful whether it belongs here at all.

b. Secondary degeneration of the spinal
the cord itself.—In all severe diseases of the
verse myelitis, myelitis from compression, hæ
sis, tumors, etc.), as we have already stated i
this book, if these diseases involve the entire
of the cord, or also certain definite portions o
ary degeneration of definite divisions of the c

If the disease involves the entire transve
least the greater portion thereof, ascending as
degeneration will be found as soon as the aff
certain length of time.

Ascending degeneration extends: *a*, to the
and especially to the inner halves of the same
per division of the spinal cord, are more sh
the outer halves, and are generally known
"tender columns," or "Goll's columns."
neighborhood of the lesion the degeneration e
width of the posterior columns, but it very so

limited to Goll's columns, diminishes in extent as it ascends, but can always be followed into the restiform bodies, where it ends. This form of ascending degeneration is very frequent, and is generally easy to recognize by its sharp limitation, and usually very distinct gray discoloration.

β. Ascending degeneration, again, may occupy *a narrow zone at the outer periphery of the posterior lateral column*, which begins with its greatest width at the point of the gray posterior horn, and extends forward over the greater part of the periphery of the lateral column, as a border which is all the while growing narrower. This degenerated zone can be followed upward to the cerebellum. It was known, although but imperfectly, to Tuerck, was afterwards often overlooked, and has recently again been restored to its legitimate place, and more carefully studied by Flechsig and Schiefferdecker. The tract thus degenerated, which closely and intimately touches the descending degenerated pyramidal tract of the lateral column, is designated by Flechsig as the "direct cerebellar tract of the lateral column" ("directer Kleinhirn-Seitenstrangband").

Descending degeneration in affections of the spinal cord also extends almost exclusively to the "pyramidal tracts," just in the same manner as in diseases of the brain, that is, with variable distribution to the tracts in the posterior lateral columns and the tracts in the internal anterior columns, only with the non-essential difference that here the gray degenerated spot in the lateral column usually has a somewhat larger extent. Naturally in spinal affections the descending as well as the ascending degeneration is generally bilateral.

Aside from this, Schiefferdecker states that transverse section of the anterior and lateral columns shows scattered fibres of descending degeneration, but they are only to be found in the neighborhood of the point of lesion.

In lesions which only affect a portion of the transverse section of the cord, we shall also find only correspondingly partial secondary degenerations.

Thus, for example, in *traumatic unilateral lesions*, the ascending as well as the descending degeneration is confined to the side of the injury.

In affections that are confined to the *fundamental bundles* *the posterior columns* ("Keilstränge"—"bandelettes externes we meet only with ascending degeneration of Goll's colum (perhaps also of the cerebellar tracts of the lateral columns), condition which probably exists with tolerable regularity in tab dorsalis.

In case of partial lesion of the lateral columns (a circu scribed sclerosis, effusion of blood, wound, tumor, etc.), we sh find only descending degeneration of the lateral column of th side.

It has not yet been determined whether certain lesions of t *gray substance* can also call forth secondary degenerations, a so far as we know the question has not yet been very close investigated. It would doubtless be worth the while more ca fully to test the anatomical conditions, with reference to t point, in amyotrophic lateral sclerosis, in the spinal paralysis children, in progressive muscular atrophy, etc.

c. Secondary degeneration of the spinal cord in disease the peripheral nerves. Such a condition is, thus far, known wi certainty to exist only in lesions of the spinal nerve-roots, an indeed, it would appear, only of the *posterior roots.* A certa number of observations exists (Cornil, Th. Simon, C. Lan Leyden) in which tumors on the cauda equina compressed t nerve-roots lying within the same without directly injuring t spinal cord itself, and in which an exquisite ascending degene tion of the posterior columns was found, extending to the enti transverse diameter of the same in the lumbar region, and bei limited, higher up, to Goll's columns.

Lesion of the motor roots, or lesion (section, crushing, etc.) the peripheral nerve-trunks is not followed by any secondary d generation in the cord.

After having thus enumerated the causes of secondary dege erations in the spinal cord, we have to add that the pathogenes of the same is by no means, as yet, satisfactorily cleared up.

Closer observation teaches that *descending* degeneration o curs in undoubtedly motor pyramidal tracts, that is, in tho leading in a centrifugal direction, only when conduction in the tracts is interrupted at some point of their course.

Furthermore, that *ascending* degeneration occurs when the posterior roots—therefore, tracts leading in an undoubtedly centripetal direction—are interrupted in their conduction; likewise, if the posterior columns, which are probably to be regarded as in great part leading in a centripetal direction, are injured; and finally, if the same thing happens to certain tracts of the lateral columns (the direct cerebellar tracts of the lateral columns). Whether the latter lead in a centrifugal or centripetal direction is not yet determined; at all events, this is not indicated with entire certainty by the direction in which these tracts degenerate on the interruption of conduction.

Some authors, then, explain secondary degenerations simply as follows: that the degeneration follows in the direction in which the conduction of physiological excitation occurs, and that the interruption of these processes of conduction—the functional inactivity of the nerve-tracts—is the actual cause of the degeneration. Therefore the motor tracts have always been found degenerated in a centrifugal, the sensitive always in a centripetal direction.

If this were true, then, when a peripheral nerve was cut through, the sensitive fibres ought to degenerate only in a centripetal direction, that is, in the central piece, the motor fibres only in a centrifugal direction, that is, in the peripheral piece. But this is notoriously not the case, both kinds of fibres degenerating in a centrifugal direction and *only* in the peripheral division of the nerve.

If this were true, then in every descending secondary degeneration of the pyramidal tracts, in consequence of lesion of the brain or cord, the anterior roots and the motor peripheral nerves would have to degenerate, for these, too, are entirely disturbed in their function. The contrary, however, is true; the anterior roots and the peripheral motor tracts remain intact in the vast majority of cases (if no special complications exist).

The view, therefore, that the degeneration is due to the functional inactivity cannot be looked upon as sufficiently well founded, as has, indeed, been satisfactorily proven by Bouchard.

In my opinion, the only conclusion which can thus far be drawn is, *that secondary degeneration occurs as soon as certain* *definite conduction tracts are separated from their trophic c* *tres, their centres of nutrition* (Bouchard, C. Lange, and othe

Where these centres (centres of development? Flechsig) and how they are related to the different tracts, is, to be sure, yet tolerably unknown. There are only some points in reg to which we can thus far draw conclusions with more or less pr ability of being right; thus, for instance, that the trophic cent for the sensitive tracts ascending with the posterior roots (the fore, probably, also for the greater part of the tracts subject ascending degeneration) are probably situated in the spi ganglia; that the trophic centres for the anterior roots and motor nerves are certainly to be sought in the gray anterior hor But it is as yet entirely unknown where the trophic centres the pyramidal tracts lie; they may, perhaps, be situated in gray cortex of the brain, in which, according to recent investi tions, large, multipolar ganglion-cells have also been found, su as exist in the gray anterior horns of the cord. The trophic c tres for the direct cerebellar tracts of the lateral columns are a as yet entirely unknown.

Consequently quite an array of important problems still mains to be solved. And however great the advantages wh have already accrued to our knowledge of the anatomical str ture and physiological functions of the spinal cord, through study of secondary degenerations, it is equally to be desired t still further and particularly experimental investigations on t highly important question should be undertaken.

Those changes in the spinal cord which have been seen to arise after *amputa* and *the resection of nerves* only stand in a very distant relation to actual secon degenerations. After amputation, a non-symmetrical condition of the spinal has been found, with narrowing of the entire half of the cord on the side of amputation, sometimes mostly affecting the anterior, and sometimes the post column, and generally also involving the gray substance. Some observers re slight histological changes, diminution in size of the ganglion-cells and nerve-fi very slight increase of connective tissue, possibly some diminution in the numb the ganglion-cells; but in the majority of instances the histological condition o spinal cord was quite normal. The asymmetry was generally more marked in portion to the youth of the individual at the time of the operation, and the le of time that had elapsed since the operation. No doubt here it is a question in of an arrest of development through the early disuse of the part; probably pa also, of simple atrophy from the same cause; and, least of all, also, of the results of a chronic irritative process travelling along the nerves from the point of amputation upward into the spinal cord. All this requires still further investigation. Thus much, however, appears to be certain already, that these changes in the cord arising after amputations, etc., have nothing to do with the secondary degenerations we are here considering; also that they are of very little practical significance.

An entirely different matter is the condition of things which Hayem found in the spinal cord of squirrels after tearing out the sciatic nerve. Here it is a question of a direct wounding of the spinal cord, and of a traumatic myelitis arising thence, the more careful study of which promises to be of great importance to the doctrine of acute central myelitis. Exceptionally such a central, ascending myelitis (which leads to progressive atrophy of the muscles) also arises after simple resection of the trunk of the sciatic nerve in the squirrel.

Pathological Anatomy.

The *macroscopic appearance* of secondarily degenerated portions is, in general, that of gray degeneration, more or less distinct and pronounced; gray or grayish-yellow discoloration sometimes disseminated with fine, whitish lines (vessels with an abundance of granule-cells). In more recent cases, a brilliant, pure, milk-white color is also to be recognized (C. Lange). Not seldom, however, in the fresh preparation, nothing at all, or nothing very definite, can be seen with the naked eye; and thus, in a fresh specimen of the cord, no correct idea can be obtained of the existence and extent of the secondary degeneration. This is generally brought out with great distinctness by the familiar coloration of the specimen with chromic acid (a bright yellow appearance), and can then be very well recognized; but a truly accurate conclusion, especially with reference to the extent of the degeneration, can, after all, only be reached by microscopic examination.

After the affection has lasted a considerable time, there is generally no trouble in recognizing distinct atrophy and shrivelling of the degenerated columns; the form of a transverse section of the cord may appear somewhat altered thereby, the gray horns being somewhat displaced in one direction or another. In one-sided degeneration, particularly, the asymmetry of the two halves of the spinal cord, on transverse section, thus stands out distinctly.

The *microscopic conditions* have not yet been develop with the completeness which we might wish, in all the differe stages of the degenerative process, so that a complete accou of the histological processes in the same cannot, as yet, given with certainty. The statements and views of differe investigators, with regard to the method and the essenti character of the process, also differ as yet in a manner that not pleasing.

In considering them, we must separate the *earlier* stag (which are far from being satisfactorily investigated as yet) fro the later stages (which come under observation by far the mo frequently).

In the *earlier stages* of the process we meet principally a almost exclusively with *alterations in the nerve-fibres.* The medullary sheaths are found strikingly altered in their reacti with carmine and osmic acid; they break down and becom fissured, suffer fatty degeneration, and finally disappear alt gether. For some time the axis-cylinders alone remain (or pe haps also the horny sheaths recently discovered by Kuehne a Ewald). Along with these processes of degeneration an excee ingly abundant development of granule-cells also sets in, whic gives a characteristic appearance to the microscopic picture, a which formerly served exclusively for the recognition of secon ary degeneration. During all this the *connective tissue is n materially increased;* this does not occur until much later. O the *walls of the vessels* an abundant deposition of fat-granul and fatty degeneration may be recognized. Aside from thi there is always a not inconsiderable number of well-preserv and apparently *normal nerve-fibres* to be observed.

In the *later stages* the degenerated tissue appears shrunke as a whole; most of the *nerve-fibres* have *entirely disappeare* or their remnants alone can still be demonstrated as narro rigid fibres (axis-cylinders? horny sheaths?). The *tissue o the neuroglia is materially increased*, and is changed into dense, finely fibrillar tissue, which contains numerous nuclei an spider-like cells. The *granule-cells have become more scant* or have entirely disappeared; not rarely numerous corpora am lacea can be seen. In this entirely degenerated tissue, likewis single, more or less numerous, well-preserved nerve-fibres are still to be seen.

It is evident, then, that these later stages of the process are in no way to be distinguished from ordinary gray degeneration (in tabes, multiple sclerosis, chronic myelitis); the histological picture is almost exactly the same. Whether this is also the case in the earlier and earliest stages, we must leave undecided, as no investigations especially directed to this point exist; indeed, the two processes (secondary degeneration and primary sclerosis) have certainly often been confounded with one another. We should think that quite considerable differences would certainly appear, and we consider it as one of the most important tasks devolving upon the students of the pathological anatomy of the spinal cord, finally to clear up these fundamental questions.

Not until this is done shall we be able to give a decided opinion as to the *method* and the *essence* of this degenerative process, with regard to which the views of different authors at present materially differ. By some the entire process is regarded as an irritative or inflammatory one, which propagates itself directly from the point of lesion by definite tracts, or which is developed in these very tracts by the breaking down of the nerve-fibres. Almost all the facts in the case argue against the theory of an inflammatory process propagated directly from the point of lesion; and if the more recent statements of careful observers are correct, that the first changes are to be recognized exclusively in the nerve-fibres, and that alterations in the connective tissue do not occur until many weeks later, then it will be impossible to sustain this view. After comparing all the statements now before us, it appears to us most probable that we have here to deal with a process entirely analogous to those changes which arise in peripheral nerves after they have been divided; that secondary degeneration, therefore, is nothing else than *the degenerative* (neurotic) *atrophy of certain definite tracts of the spinal cord separated from their nutritive centres.* We shall have to leave it undecided for the present, whether the proliferation of connective tissue which arises later is called forth by the irritation caused by the products of degeneration of the nerve-fibres, or whether it constitutes an essential part of tl degenerative atrophy.

Statements differ materially with regard to the period witl which secondary degeneration is developed after the occurrei of a lesion which causes it. Tuerck's opinion, that about hal year is required for this purpose, is evidently incorrect; all m recent observations have approximated the end of the peri much nearer to the occurrence of the causative lesion, and cases falling under this head we may be sure of finding t degeneration distinctly developed and easily recognizable af four, six, or eight weeks. Schiefferdecker, in his experime on dogs, found the beginning of the degeneration at the end fourteen days. At the end of four to five weeks the well-mark picture of the same was completed, and it was not until t eighth week that he saw changes arise in the connective tissue.

The peculiar form of degeneration in ring-shaped figures, which Westp described, and which was caused by a massive, peculiarly localized accumulat of granule-cells, has not been seen by others, and its significance is as yet entir unknown. C. Lange thinks it was not a case of secondary degeneration, bu peculiar form of myelitic alteration; perhaps it belongs under the head of w Schiefferdecker calls "traumatic degeneration."

The *distribution, as to space*, of secondary degeneration the spinal cord is, in almost all cases, strictly according to ru and extremely characteristic.

Descending degeneration spreads first, in all cases, to the p terior half of the lateral columns—not entirely, but in grea part filling out the space between the periphery, the posteri gray horn, and the region near the point of attachment of t ligamentum denticulatum. On making various transverse s tions, the transverse picture of the degeneration produced somewhat varied (see Fig. 18).

In the secondary degeneration which follows diseases of t brain, the degenerated portion, at least in the upper half of t spinal cord, does not reach the periphery of the lateral colum but remains separated from the pia mater by a narrow margin sound tissue, and is generally also somewhat separated in t same way from the limits of the posterior gray horn. Furth downward, on the other hand, this healthy peripheral marg disappears, and the degeneration, although, as a whole, it has grown smaller, reaches entirely to the pia mater in the lower dorsal and lumbar portions of the cord.

The secondary degeneration in such cases can generally be first demonstrated in the medullary substance of the crus cerebri; then in the pyramid of the same side, to be followed through the crossing of the pyramids into the lateral column of the opposite side; the greater part of the degenerated fibres takes this course, while a smaller part often remains on the side of the lesion, to continue its course downward in the pyramidal tract of the anterior column of the same side.

The degeneration of the lateral column is most extensive in the cervical portion, and here has more of a triangular form, with the apex directed inward (Fig. 18, 1 to 3); in the dorsal portion it becomes gradually narrow and assumes more of a roundish form (Fig. 18, 4 and 5); in the lumbar portion it moves more towards the periphery, again assumes more of a triangular form, grows narrower and narrower, finally to disappear entirely at the height of the origin of the third or fourth sacral nerve (Fig. 18, 6 to 8).

Aside from this, in descending degeneration, the inner surface of the anterior column (pyramidal tract of the anterior column) on the same side with the cerebral lesion is usually found degenerated to a greater or less extent. It constitutes a narrow zone, which generally does not extend as far down the cord as the degeneration of the lateral column, but disappears entirely (Fig. 18, 1 to 6). The occurrence of this degeneration depends on the distribution of the pyramidal tracts in the transverse

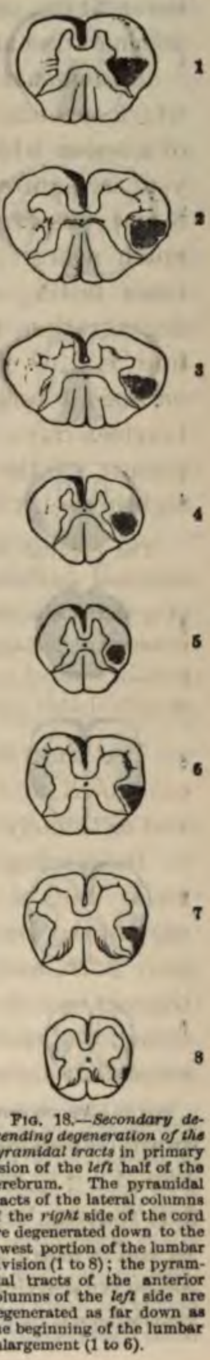

FIG. 18.—*Secondary descending degeneration of the pyramidal tracts* in primary lesion of the *left* half of the cerebrum. The pyramidal tracts of the lateral columns of the *right* side of the cord are degenerated down to the lowest portion of the lumbar division (1 to 8); the pyramidal tracts of the anterior columns of the *left* side are degenerated as far down as the beginning of the lumbar enlargement (1 to 6).

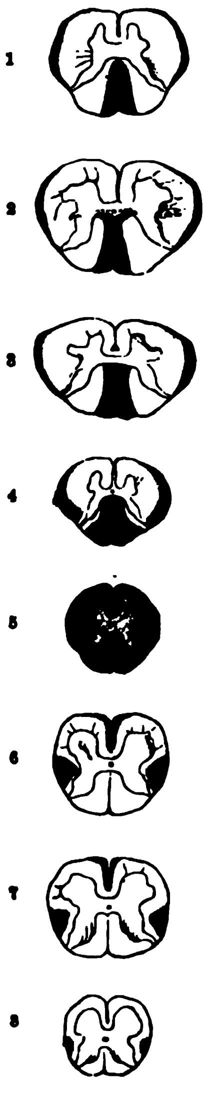

FIG. 19.—*Myelitis transversa dorsalis, with secondary ascending and descending degenerations* (half schematic). Seat of the lesion at the height of section 5. Above this (1 to 4), secondary *ascending* degeneration in Goll's columns and the direct cerebellar tracts of the lateral columns. Below the lesion (6 to 8), secondary *descending* degeneration in the pyramidal tracts of the lateral and anterior columns.

diameter of the spinal cord, as has been more carefully investigated by Flechsig; *only* the pyramidal tracts degenerate. Accordingly, in one-third of all the cases the lateral column alone is found degenerated; in the remaining two-thirds the degeneration also involves the anterior column of the opposite side. Thus far no instance has been found of degeneration of the pyramidal tract of the anterior column alone.

In diseases of the spinal cord the relations of descending degeneration are precisely the same. Only the transition into the original focus of disease is usually very gradual, so that the secondary degeneration is only separable, little by little, from the more diffuse lesion of the spinal cord. Here the degeneration everywhere reaches the surface of the cord and can generally be recognized glimmering through the pia mater (Charcot, C. Lange). The distribution to the lateral and anterior columns is the same as in diseases of the brain (Fig. 19, 6 to 8). Naturally, in affections of the spinal cord the degeneration is generally bilateral; still it may also be the same in bilateral brain affections.

Ascending degeneration of the posterior columns involves that portion of the posterior columns lying next to the posterior fissure, usually having a very sharp outline (Fig. 19, 1 to 4). In the upper portions of the cord it is strictly confined to the columns of Goll, its apex usually not quite reaching the posterior commissure. In the neighborhood of the original focus of disease the degeneration spreads over the entire transverse diameter of the posterior columns, thus gradually to pass over into the diffuse lesion.

Above, the ascending degeneration extends into the medulla oblongata and ends here with the "Funiculi graciles" (of Burdach—"zarten Stränge" of Erb), in the region of the nuclei of the same.

In tumors of the cauda equina with secondary degeneration, the posterior columns are degenerated to their entire width, in the lower division of the spinal cord; the degenerated zone grows gradually narrower and is finally strictly limited to the columns of Goll (Th. Simon, C. Lange). This diminution, according to Schiefferdecker, does not occur progressively and uniformly, but by steps.

Ascending degeneration in the lateral columns is liable to occupy but a very insignificant amount of space. It appears in the form of a narrow peripheral zone, not sharply defined towards the inside, which begins at the hindmost end of the lateral column and extends around the periphery of the same to a greater or less distance forward—often to the neighborhood of the anterior columns (Fig. 19, 1 to 4). Below, this degenerated zone arises gradually from the more diffuse lesion of the spinal cord; farther up it can always be traced on the outermost periphery of the cord in the restiform bodies and until the entrance of the same into the cerebellum. Schiefferdecker states that this secondary degeneration also diminishes by steps.

Secondary degenerations, in the vast majority of cases—in fact we may say regularly—confine themselves to the white columns of the cord. The gray matter generally remains entirely intact (Tuerck, Bouchard), and but very exceptionally takes part in the process. Nevertheless, in descending degeneration of the lateral columns a spread of the (irritative ?) process to the gray anterior horns has repeatedly been observed (Charcot, Pierres, Pitres), resulting in degeneration of the large ganglion-cells and muscular atrophy. This has thus far been observed principally in the cervical enlargement, but belongs, as above stated, to the rare exceptions.

Symptomatology.

Ascending secondary degeneration (in the posterior column and in the cerebellar tracts of the lateral columns), so far as w are at present informed, appears to occasion no sort of clinica manifestations; that is, it adds no new disturbances to thos caused by the fundamental malady. At the same time the que tion might be raised, whether the manifestations of sensory irri tation in the paralyzed side of the body (paræsthesia, formica tion, pain, a sensation of burning, of heat and cold, etc.), whic arise so often in the myelitis of compression and similar condi tions, may not be connected with ascending degeneration of th posterior columns, and originate and be maintained thereb This does not appear to us so entirely improbable, although i might, indeed, be hard to prove.

However positively, therefore, we may expect the occurrenc of secondary ascending degeneration in certain affections of th spinal cord when their diagnosis is once fairly established, an however regularly we also find evidence thereof on the dea body, yet this certainty is by no means founded upon any def nite clinical symptoms; it rests, the rather, upon constant path logico-anatomical experience.

It is somewhat different with the *descending degeneration* *the pyramidal tracts.* Of course this can produce no change i the phenomena of paralysis caused by the original malady. B since the work of Bouchard it has become to a high degree prob ble that the *slowly developed contracture of hemiplegic subjec* (the "contracture tardive" of the French), which becomes ass ciated with the paralysis at a later period, stands in connectio with the secondary degeneration of the pyramidal tracts, and the result of the same. Bouchard is of the opinion that it is n so much the degeneration of the tracts directly affected by t paralysis which causes the contracture, as the irritation of oth nerve-fibres, of different origin, running in the same region of t cord, which irritation is caused by a sclerosis arising in cons quence of the degeneration. This will, at all events, be a ha matter to prove; and, indeed, it is not easy, as yet, to for muscular tensions and contractures set in after some weeks, we may infer, with great probability, the existence of descending degeneration of the pyramidal tracts.

If this should be confirmed in the future, then the demonstration of the presence or absence of this secondary degeneration will perhaps admit of our arriving at a conclusion with regard to the localization of the fundamental cerebral affection. For, as we have before remarked, this degeneration does not by any means occur in all, but only in very definitely localized affections of the motor portions of the brain (Charcot). This is a question that will have to be decided by cerebral pathology.

Prognosis.

The more exact history of the course of secondary degenerations, under various possible circumstances, is a matter yet to be investigated.

In general, the prognosis is the same as that of the fundamental affection, and we have every reason to believe that, with the cure of the fundamental affection, the secondary degenerations may also tend towards resolution and recovery. But this is not so very certain under all circumstances, and although a complete regeneration of the degenerated nerve-tracts appears possible in the earlier stages of the process (similar to what occurs in the degenerative atrophy of peripheral nerves), yet this is, to say the least, improbable in the later stages of the affection, and the prognosis of secondary degenerations, after they have existed for a long time, is, therefore, to be regarded as decidedly unfavorable.

Naturally, under some circumstances, the prognosis of the fundamental malady is seriously vitiated by this means, and Charcot unhesitatingly regards descending degeneration of the pyramidal tracts as one of the main causes of the persistence of motor paresis in hemiplegia, even when the fundamental affection runs a favorable course. There is furthermore no doubt that secondary contractures often cause much greater interference with the use of the extremities than would result from the degree of paresis or paralysis actually present. And if these arise without them, etc. On account of the short time that ha elapsed since the discovery of the reflex excitability of tendons such observations do not exist in sufficient numbers. I only fin some statements in Westphal,[1] that after apoplexies and the lik the elevation of reflex excitability of the tendons is present i from seven to twenty-one days after the attack, and in som cases was not present during the first four days. But we ca not form much of a conclusion from this, as the statements wit regard to the time of occurrence of secondary degeneration a as yet very uncertain. It appears to me, for the present, th more probable that the elevation in reflex excitability of th tendons is the result of interrupted conduction in tracts controlling reflex action, and is not the result of secondary degeneratio of the pyramidal tracts. At all events, this question demand further investigation.

It is at least not likely that the epileptic and epileptifor attacks, which are so frequent in hemiplegic patients, have an relation whatever with secondary degeneration of the pyramid tracts (especially in the medulla oblongata), as was supposed b Bouchard.

Diagnosis.

From all that has hitherto been said, it is evident upon ho unsafe a footing the clinical diagnosis of secondary degeneration as yet stands.

Secondary *ascending* degeneration is simply not to be recognized by any clinical manifestations; but, in case of the well established diagnosis of a definitely localized affection of th spinal cord (myelitis from compression, trauma, etc.), it is merel to be expected, with some certainty, as a necessary result of th same.

In secondary *descending* degeneration the only sign which points towards it with some probability is the occurrence of lat contractures (and of increased reflex excitability of tendons?). When, therefore, in the course of a hemiplegia or paraplegia, muscular tensions and contractures set in after some weeks, we may infer, with great probability, the existence of descending degeneration of the pyramidal tracts.

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[1] Arch. f. Psych. u. Nervenkrankh. V. p. 810.

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All this will therefore have to be taken into consideration in forming the prognosis of the cerebral or spinal fundamental affection.

Therapeutics.

There has thus far not been much talk of any special therapeutics of secondary degenerations; indeed, this could not well be the case, in view of the difficulties which stood in the way of the diagnosis of these alterations.

The essential point, of course, is the treatment of the fundamental affection, with regard to which we need not here expatiate.

But after what has been said above, the question may well be raised whether a further and not inconsiderable improvement might not be attained through direct treatment of the secondary degenerations at a certain stage of the fundamental malady; whether, after the causative cerebral malady had, to a great degree, undergone resolution, and after cerebral conduction had again been restored by direct or by vicarious tracts, the paresis and contractures which still remained might not be removed by the removal of the secondary degeneration.

This question may doubtless be answered, with probability, in the affirmative; and *it therefore appears to be indicated, when the fundamental disease has improved and is in the act of retrograde development, to institute direct treatment of the secondary degenerations.*

The ways and means to this end are, it is true, yet to be found. *A priori*, one will feel inclined to resort to the employment of the therapeutic measures commonly used in chronic myelitis and in sclerosis in general (baths, cold-water cures, galvanism, etc.). The *galvanic current* is doubtless entitled to the most confidence, and admits of the most practical and safest method of application. It is not necessary to say anything further here with regard to the methods of its application in these degenerations of the cord. I will merely add that in some old cases of hemiplegia with contractures I believe I have seen decidedly favorable effects on paresis and contractures follow direct galvanic treatment to the spinal cord, instituted in addition to the other galvanic treatment. In such cases, however, it is naturally very difficult to be sure about our therapeutic experiences.

20. *Deformities and Malformations of the Spinal Cord.*

Ollivier, l. c. 3. éd. I. p. 159–240. 1837.—*Cruveilhier*, Anatom. patholog. Livrais. XVI.—*Virchow*, Die krankhaften Geschwülste. I. S. 169. 1863.—*Foerster*, Handb. d. path. Anat. 2. Aufl. II. S. 621. 1863.—*P. Schiefferdecker*, Asymmetrie d. grauen Subst. des R.-M. Arch. f. mikrosk. Anat. XII. S. 87. 1876.

Bardeleben, Lehrb. d. Chirurg. u. Operationslehre. Bd. IV.—*Lorinser*, Spina bifida in *Pitha-Billroth*, Chirurgie. Bd. III. 2. S. 5.—*Smith*, An Unusual Form of Spina Bifida. Transact. of the Pathol. Soc. XXI. p. 1. 1871.—*J. Roose*, A Case of Spina Bifida. Philad. Med. Surg. Report. 1872. March 30.—*A. Petit*, Analyse d'un liquide de spina bifida. Bull. génér. de thérap. 1873. p. 256.—*Th. Simon*, Ueber Syringomyelie und Geschwulstbildung im R.-M. Arch. f. Psych. u. Nervenkrankh. V. S. 121. 1874.—*Leyden*, Klin. d. Rückenmarkskrankh. I. S. 195. 1874; Hydromyelus u. Syringomyelie. Virch. Arch. Bd. 68. S. 1. 1876.

We have but little to say in this section that is of interest to the practical physician and really necessary for him to know.

First in order comes quite a series of things which are of interest only in a pathologico-anatomical point of view and in connection with the history of development; and which, to the practical physician who is primarily concerned with living humanity, will prove all the less interesting because they occur only in the still-born fœtus, or at least in one incapable of sustaining life.

On the other hand, there are certain anomalies in the configuration and position of individual divisions of the spinal cord which one must know in order to avoid the possibility of confounding them with pathological changes. This also is a matter which more particularly interests the pathological anatomist or the special investigator of the pathology of the spinal cord. The practical physician will but seldom obtain a sight of these things, which do not betray themselves by any symptoms during life.

Finally, we have to deal with conditions which are congenita are carried into later life, and are perhaps still further develope at a later period—conditions of extension and widening of th cavities lying within the spinal column itself or surrounding th same, with more or less considerable accumulations of flui within the same and corresponding degrees of secondary change —displacements, adhesions, the development of clefts of th cord itself, or of the parts surrounding it (hydromyelus, hydro rhachis, and spina bifida). These things, being, in part at leas forms of disease susceptible of clinical diagnosis and therapeuti interference, must be taken up and discussed. But, in view o the pre-eminently surgical interest which attaches to the mos important of these anomalies—spina bifida—we may be tolerabl brief in its discussion.

Among the first group of anomalies, as mentioned above, ar the following :

1. *Amyelia*, or *absence* of the *spinal cord*. This is a com paratively rare malformation. Ollivier counts up a series o older observations of the same. If the spinal cord is missin then the brain is always missing too (amyelencephalia). Enti absence of the spinal cord, when the brain is present, does n seem to occur. The peripheral nerves may be entirely preserve the nerve-roots partly so ; remnants of the spinal membran may also sometimes be found in connection with rudimentar development of vertebræ. It is not necessary here to enter upo any more minute description.

It is also stated that during the latter months of pregnanc the greater part of the spinal cord may be destroyed by hydr myelus—the brain, as well as the peripheral nervous system, bein maintained intact. This, too, is of no practical significance. A such products of generation are incapable of life.

2. *Atelomyelia, or imperfect development of the spinal cord.*

The upper end of the spinal cord is lacking, or is imperfectl developed, in all cases in which the brain is lacking (*anenc phalia*), or in which the greater part of the head is defecti (*acephalia*). The medulla oblongata is then lacking, or exis only in a rudimentary form, sometimes still in conection with part of the nerves usually given off from it. Farther down, th development of the spinal cord may then be quite regular. Sometimes, however—in acephalia—a larger piece of the upper division of the cord is entirely wanting ; in that case there is also usually spina bifida of the cervical vertebral column.

In rare instances the lower part of the spinal cord is also found stunted, ending in a club-shaped extremity ; this is generally accompanied by still further anomalies of the organs of the trunk.

The interference with development may also show itself in the fact that the two lateral halves in which the spinal cord is developed do not unite, but either remain entirely separated, or unite only throughout a portion of their extent; so that the spinal cord, either for its entire length or merely at its upper or lower end, appears separated into two lateral halves—*diastematomyelia.* This malformation generally occurs with anencephalia, and is, of course, of no practical significance.

3. *Diplomyelia*, or *duplication of the spinal cord.*

Double monstrosities of various kinds and of various grades also show these malformations of the spinal cord, corresponding to the rest of their bodily development; so that either the upper part of the spinal cord is single and is divided into two cords below, the vertebral column and the entire lower half of the body also being double, or the lower part of the cord is single and terminates in a double cord above.

We are not yet in possession of more accurate information with regard to the method of duplication or the connection of the parts at the point of bifurcation, which would certainly afford very interesting pictures on transverse section.

In such cases the form of the spinal cord probably has but little influence on the entire development and life-history of these monsters.

Turning to our second group, we may first mention those *slight anomalies in the length and thickness of the cord* which are now and then met with. In some men the spinal cord is found strikingly thick and voluminous, in others strikingly thin and insignificant, without either the one class or the other having presented any spinal symptoms during life. If, at the same time, microscopic examination demonstrates no alteration in the structure of the spinal cord, then we are evidently dealing with irrelevant, individual anomalies.

This is regularly the case with *abnormal length* (the cord has been seen to descend to the third lumbar vertebra) or *abnormal shortness* of the cord (sometimes it ends at the eleventh or twelfth dorsal vertebra), if there are no other special malformations or diseases present.

Abnormal smallness of the entire spinal cord and medulla oblongata, with corresponding smallness of the nerve-cells and fineness of the nerve-fibres and axis-cylinders, has lately been demonstrated by F. Schultze,[1] in one of Friedreich's cases of "hereditary ataxy," and he propounds the interesting query, whether this faulty development of the entire organ is not the predisposing cause of the sclerosis of the spinal cord developed at the time of puberty. This question would be well worth testing in future cases.

Those alterations which sometimes show themselves in the *configuration* of the spinal cord, and which must not be confounded with pathological changes, with actual diseases, are more noticeable.

This is especially true of *asymmetry of the gray substance;* unequal width, depth and form of the symmetrical gray horns, on a transverse section. Starting with the supposition that the symmetry of these parts was very complete, some observers have often declared deviations from the same to be pathological; but this is not permissible, unless accurate histological examination demonstrates actual pathological changes which account for such asymmetry (atrophy, sclerosis, the loss of ganglion-cells, etc.). Schiefferdecker has demonstrated that such asymmetry is not rare, occurring even to a very considerable degree, and may be confined to single divisions of the spinal cord; the same thing was stated long ago by other observers, and every one who occupies himself with the histology of the spinal cord will often meet with this fact.

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[1] *Friedreich*, Ueber Ataxie mit besonderer Berücksichtigung, u. s. w. Nachtrag. Virchow's Archiv. Vol. 70. p. 140. 1877.

The non-uniform distribution of the pyramidal tracts upon the two sides of the spinal cord, as demonstrated by Flechsig,[1] whereby each pyramid may send its mass of fibres into the spinal cord either entirely crossed or only partly crossed, sometimes leads to very pronounced asymmetry, which here affects the white substance exclusively, and, indeed, the antero-lateral columns.

In cases of congenital absence or intra-uterine stunting of certain extremities, we not rarely see secondary atrophy of definite portions of the spinal cord, and thereby the production of asymmetry which is limited to the cervical or lumbar enlargement, according to the extremity that is affected. Thus Troisier,[2] for example, found a unilateral diminution in size of the spinal cord especially affecting the gray matter, running through the entire extent of the cervical enlargement, in a case of congenital stunting of the corresponding upper extremity (hemimyelia). In this case, without any further demonstrable changes, the only thing that could be shown was a diminution in the number of the ganglion-cells.

Unusual outgrowths of the gray matter, or the absence of portions of the same, which are generally present (for instance, the *tractus intermedio-lateralis* of Clark's columns), are conditions occasionally met with; likewise, in some cases, duplications of the one or the other gray horn, for a longer or shorter distance. These are matters the true value of which we can generally arrive at, without much difficulty, on careful investigation.

In the third group we turn our attention first to *congenital enlargement of the central canal in the spinal cord*, what is generally called *hydrorrhachis interna*, also hydromyelus (or better, hydromyelus congenitus).

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[1] Die Leitungsbahnen im Gehirn und Rückenmark des Menschen. Leipzig, 1876.—Ueber Systemerkrankungen im Rückenmark. Archiv der Heilkunde. XVIII. 1877.

[2] Arch. de Physiologie. IV. p. 72. 1871–'72.

This anomaly is not so very rare; but by no means all tho cases in which this condition has been thought to exist hav been actual congenital malformations; they have oftener repr sented changes acquired at a later period, as the result of diseas and more frequently yet these have not been cases of dilatatio of the central canal itself at all, but of newly-formed patholog cal cavities—cavities which have only been confounded with tru hydromyelus on account of their central position in the spin cord, and which would be better designated "syringomyelia. This subject will be touched on again at greater length, in th following section (No. 21).

The malformation which concerns us here depends on a *accumulation of fluid in the fœtal central canal.* This ma reach very varied grades.

In the *lighter grades* we recognize the formation of a centr cavity in the spinal cord, more or less extensive in length, an varying in width from that of a knitting-needle to that of a crow quill. This may extend throughout a greater or less portion of th cord, sometimes running through the entire length of the sam at other times restricted to certain portions thereof (the cervic or lumbar portion); sometimes the dilatation assumes the for of a string of beads (moniliform), dilated portions alternatin with those of normal width; sometimes, too, the canal is oblite ated in the centre, its anterior and posterior walls having grow together, leaving both sides open, so as to produce the appearan of a double canal, etc.

In such cases, as a rule, the dilated central canal can be re ognized as such, with certainty, by the position of the cavit relative to the other portions of a transverse section of the cor (relative to the anterior and posterior commissure, to the centra veins, the gray matter, etc.), by the lack of any trace of anothe structure which might be the central canal, by the fact of it being lined with cylindrical epithelium, etc. The spinal cor itself is otherwise well developed, and appears to be withou any other pathological changes, aside from the displacemen which its other portions may have been subjected to through th great dilatation of the canal.

This is also a positive test for the separation of congenita dilatations of the central canal from those caused by pathological processes. The latter are not rare, for instance, in periependymal myelitis, in transverse myelitis, in neoplasms of the spinal cord, and we shall recur to them again later.

Simple congenital hydromyelus appears to produce no symptoms whatever; much of the observations under this head have been made accidentally, in persons who had complained of no spinal symptoms during life; and where such symptoms have existed and "hydromyelus" has been found on post-mortem examination, these pathological processes have undoubtedly, as a rule, co-operated in the production of the same.

In the *higher grades* of hydromyelus entire disappearance of the spinal cord takes place, or it appears split in two halves, for a greater or less length; the cavity of the central canal is in free communication with the cavity of the dura mater and the hydrorrhachis interna is merged into hydrorrhachis externa, as not rarely occurs in spina bifida. This condition of things, too, may be developed in very different grades and to a variable extent.

An abnormal accumulation of fluid within the cavity of the dura mater—that is, in the subarachnoidal spaces, in connection with a greater or less degree of alteration and deformity of the vertebral column, constitutes what is called

Spina bifida (partial hydrorrhachis, hydromeningocele, and hydromyelocele).

We shall have to spend a little more time on this, as being, practically, the most important malformation, although, for all more minute details, we refer our readers to the text-books on Surgery and Pathological Anatomy.

Spina bifida presents itself as a sac-like dilatation and pouching of a more or less circumscribed portion of the cavity of the dura mater, which is generally coincident with the cleaving (or absence) of one or more vertebral arches, and protrudes like a hernia through the cleft thus formed, raising the skin in the form of a more or less considerable tumor. This pouching but seldom occurs between two vertebral arches without cleaving of the same, and if it does, is always a comparatively small tumor. (If the vertebral column remains quite normal, and there is only an increased accumulation of water in the vertebral canal, then we have simple hydrorrhachis externa—a common manifestation i hydrocephalus.)

Opinions still differ as to the exact method of origin of spin bifida, and it is at least probable that this condition may t brought about in various ways. It is either a question of a pr mary dropsy of the central canal, with disappearance of the co and secondary widening and distention of the spinal membrane or merely of a dropsy of the sub-arachnoideal spaces, which di tends the membranes and pushes them forward like a hernia. these changes take place *before* the vertebral arches are close their closure is prevented, and thus the cleft of the vertebr column takes place. But it is also possible that this is the pı mary step, and that the hydrorrhachis is not developed un afterwards.

Spina bifida is not exactly a rare malformation. Chaussi found it present in about one case in every thousand births.

The more special relations of spina bifida are about as fc lows:

Its general seat is in the sacral and lumbar region, much mo rarely in the dorsal or cervical portion of the spinal colum generally there is but one tumor present, more rarely, severa these are almost always situated in the median line, seldo deviating to one side or the other.

They differ very much in size, varying from that of a nut that of a head. In *form* they are usually round or elliptica or also pear-shaped; but they may also be extended in lengt stretching over a more or less considerable portion of the enti spinal column. The tumor is either flat or pedunculated, som times lobulated or divided into two.

The *skin* over the tumor is either quite normal or highl stretched, thinned, reddened, involved in ulceration; not rarel an umbilicated depression may be seen at some point on th height of the tumor, caused by the insertion of the end of th spinal cord in the interior of the sac.

Passing beneath the skin, one usually comes upon the dur mater, which may be thickened or normal, or also very muc thinned; sometimes it has entirely disappeared or has split, an the soft membranes alone protrude. Next comes the arachnoid which, in most instances, actually encloses the fluid. If hydromyelus exists, the pia mater also takes part in the formation of the sac.

The neck of the sac is more or less narrow and leads into the spinal canal. Not unfrequently it is closed by adhesion, and thus the cystic tumor lying under the skin is isolated from the cavity of the dura mater within the vertebral canal.

The condition of the spinal cord varies in individual cases. In the majority of instances the spinal cord is certainly essentially normal, and takes no part, or but a very subordinate part, in the existing alterations. It happens still more frequently that its lower extremity is adherent to the sac at some point, and causes an umbilicated depression of the same, visible from without. The spinal cord is often materially lengthened by this means, its extremity being drawn clear out of the vertebral canal, and not seldom thinned and flattened. The nerve-roots then run their course from this portion back along the walls of the sac (or floating free within the same), partly into the vertebral canal and to their intervertebral foramina, partly directly through the walls of the sac to their appropriate plexus. More rarely there exists simultaneously a hydrorrhachis interna, with its evil consequences so far as regards the form of the cord; then the lower extremity of the cord is more or less destroyed, atrophied, and the cavity of the sac communicates directly with the dilated and open central canal.

The *fluid* contained in the spina bifida is generally quite light, colorless, and clear, and perfectly identical with the usual cerebro-spinal fluid, consequently very rich in water and poor in solid ingredients; it is but rarely turbid or bloody; its quantity may amount to from 500 to 1,000 grammes (3 oz. to 2 lbs.) or more.

Symptoms of Spina Bifida.

Children affected with this deformity are generally born without hindrance, are capable of sustaining life, and show nothing abnormal except a more or less extensive tumor in the sacral or lumbar region, rarely at a higher point of the spinal column.

If the tumor is very large and the skin over it thinned, sac may burst during birth and its contents escape; the ch is then ordinarily born highly asphyxiated, and perishes withi couple of hours, or, at the outside, in a couple of days.

If this is not the case, however, the tumor either remains changed, or, more frequently, gradually increases in size, skin over the same becoming more and more thinned, etc.

It is but rarely that such a tumor is first developed some ti after birth. See Genga's case in Ollivier.

The tumor itself, varying very much in size, is roundish, pe shaped, or divided into two parts and lobulated, tense, elas and distinctly fluctuating. In smaller tumors the skin over same is unaltered, in larger ones it is more or less stretched a thinned, bluish or reddish; at the same time the entire tun not rarely looks translucent, like a hydrocele. In worse cas the skin is superficially ulcerated and secretes a watery, purul fluid; it may in part be entirely destroyed or absent, so that distended spinal membranes alone constitute the wall of sac.

A characteristic found in many cases is that the fluid c tained in the sac can be pressed away. In other words, it c by pressure, be partly emptied into the spinal canal, so that tumor grows smaller; but this does not occur without sometin causing disturbances of cerebral innervation—drowsiness, sle twitchings, etc. If there is coincident hydrocephalus, sw ing and increasing protrusion of the fontanelles can be dem strated during such pressure; on the other hand, pressure the hydrocephalic skull increases the tension and size of spina bifida. The swelling likewise somewhat increases on suming the erect attitude, on screaming, on pressure, etc.; increase of swelling has also been observed associated with mc ments of the pulse and of respiration. Not infrequently tumor is sensitive on pressure. In those cases, however, in wh communication with the spinal canal has been cut off by clos of the sac, all these manifestations are, of course, no longe be found.

In the majority of cases the manifestations above describ are the only ones in spina bifida. The children thus affected otherwise normal, show no sort of manifestations of paralysis, develop well, learn to walk, and *may* arrive at a considerable age without being troubled by their deformity otherwise than, at most, in a simply mechanical manner.

This, however, is the exception; as a rule, the tumor advances in growth, and not long after birth all sorts of severe disturbances begin to show themselves, caused by compression of the lower division of the spinal cord and the cauda equina. Increasing paralysis and paraplegia set in, incontinence of urine and of fæces, bed-sores, etc., and the patients thus gradually perish.

Much more rarely these severe manifestations exist at the very beginning, and this is regularly the case whenever there is coexistent hydrorrhachis interna. Paraplegia and paralysis of the sphincters are then present from the very start, and the days of the little patient are numbered.

But these children by no means always perish as the direct result of the spinal paralysis thus caused; much greater and more urgent dangers threaten them from the numerous acts of violence and accidental wounds to which the tumors are exposed. Knocks, pressure, dragging, direct wounds, prolonged lying upon the back, and the like, are all of them capable of bringing about a rupture of the sac. This results in purulent inflammation of the same, which—if the communication with the spinal canal is still open—extends into the latter, and induces a purulent spinal meningitis which ordinarily puts an end to the life of the little one within a few days. It has happened, although but very rarely, that such an occurrence has passed over favorably and has even led to the cure of the malady; this is only to be expected when the perforation is very minute and the fluid flows out quite slowly. If the opening is large and the fluid is, by any accident, rapidly evacuated, death may follow very quickly, within a few hours, under general convulsions and increasing debility.

The *terminations* of the disease have been sufficiently indicated in what has just been said. Life is but rarely maintained for any considerable length of time; still, isolated cases have been known to live to the age of 20, 30, 35, and even 50 years. Prob-

ably in all these the communication with the cavity of the du
mater was closed early, or was very narrow.

In most cases death occurs much earlier, and, indeed, th
more certainly and quickly the larger the tumor. The immedia
cause of death is most frequently rupture of the sac; th
comes about either through the fact of the tumor gradually gro
ing in size, and finally bursting of itself or through some ac
dental wound; or through the fact of some traumatic injur
wound, blow, or the like, causing a sudden rupture; or throu
the fact of the sac being purposely opened and emptied for the
peutic purposes. If, in such cases, the emptying takes pla
quickly and in a stormy manner, death may occur within a fe
hours, under general convulsions and increasing weakness; or-
and this is more frequently the case, especially after operatio
—an acute purulent meningitis is developed, which leads to th
death of the child in a few days.

In a minority of instances death follows without bursting
the sac, through general prostration in consequence of the seve
spinal disturbances.

The rarest result of all is that such an accidental perforatio
is survived, and becomes the starting-point of a positive cur
More frequently, though by no means frequently enough, thi
favorable result follows in consequence of a carefully undertake
operative evacuation of the sac.

Diagnosis.

The recognition of spina bifida is generally not difficult; onl
in the smallest forms of the same, when the orifice of communi
cation is very small or entirely closed, can this present any con
siderable difficulties.

The following may be mentioned as distinguishing marks:
tumor on the vertebral column, of the form and size describe
above; tense, fluctuating, translucent; its size capable of bein
reduced by pressure, whereupon the fluid is emptied into th
spinal canal, causing the appearance of severe nervous disturb
ances (finally also protrusion of the fontanelles); the edges o
the cleft vertebræ, the ends of the vertebral arches being some

times distinguishable to the touch as light prominences. By these means spina bifida will, as a rule, be easily diagnosticated.

It is of special importance—in a prognostic point of view, as well—to demonstrate the presence or the absence of communication with the spinal canal ; this is not easily done in all cases.

The distinction between this and the displacement of abdominal and pelvic viscera (bladder, rectum, etc.) resulting in a sacral tumor, to which Lorinser calls attention, will generally be easy. There will likewise be no difficulty in recognizing the other malformations and defects which not rarely coexist with spina bifida ; viz., hydrocephalus, deformities of the lower extremities, anomalies of the genital apparatus, inversion of the bladder with congenital fissure of the abdominal walls, and the like.

Prognosis.

The prognosis of spina bifida is by no means very favorable. Very many cases perish by the spontaneous accidental opening of the sac or through the progressive growth of the tumor ; not a few, likewise, die in consequence of operations undertaken to effect a cure.

The prognosis becomes more unfavorable the larger the tumor is, as a whole ; the higher it is situated on the vertebral column (the cases where it is on the sacrum and coccyx are by far the most favorable) ; the larger the orifice of communication with the spinal canal ; the more it is a case of true myelocele, and not mere meningocele—therefore, the more there is of hydromyelus ; the more distinct the signs of hydrocephalus ; and, finally, the feebler the constitution of the child thus affected. Keeping these points in view, one will generally be able to form a conception of what is to be expected during the further course of the malady.

Therapeutics.

Nothing at all is to be expected from the treatment of spina bifida with internal remedies, with inunctions, baths, etc. The attempts to cause the disappearance of this condition by derivation to the skin, by blisters, the actual cautery, etc., may designated as downright objectionable.

But it is just as little permissible in most cases to leave thing to itself and expect time to effect a cure. Experie teaches that this trouble is almost always of a progressive ch acter, and sooner or later leads to death.

Nothing remains, therefore, but *surgical treatment,* wh must, however, be undertaken with the greatest caution a care, if the results are not to be altogether too unsatisfactory.

Very different operative measures have been undertak naturally with very various results. Lorinser is opposed to operative interference as long as a communication with the spi canal can be demonstrated. This is probably, after all, somew excessive caution, although it is evident, in and of itself, t those cases which have but a very narrow communication, none at all, with the cavity of the dura mater, promise far bet results than those with a very wide communicating orifice, as main danger of the operation is always to be found in the ext sion of an inflammatory process to the membranes of the spin

Among the operative procedures hitherto proposed, there a number that are to be positively rejected, because alm always accompanied by fatal results. First among these is *passing of a hair cord through the tumor;* this is inevitab followed by inflammation and suppuration, and could only considered reasonable if one were quite sure that the exter tumor was entirely shut off from the spinal cavity. The *incis* or *amputation of the tumor* is likewise commonly followed fatal meningitis, and is therefore to be avoided.

Ligature of the sac, which implies that it is pedunculat seems to be almost equally dangerous; the sac sloughs off, orifice of communication cicatrizes, and a cure may thus effected; but here, too, purulent meningitis, with a fatal resu very often follows. At the same time a number of favora results, following this method, have been reported. Thus, Rizzoli and Oldoini,[1] by Parona,[2] who availed himself of a p of compressing forceps for carrying out the operation; by von Brunn,[1] who undertook the compression of the pedicle by means of an ovarian clamp; by Colognese,[2] who used an elastic ligature, a rubber tube, for this purpose.

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[1] Lo Sperimentale. XXXVIII. (Centralblatt f. d. med. Wiss. 1876. No. 50.)

[2] Annal. universal. Vol. 235. Aprile, 1876.

In view of the better results attending them, the following methods of operation are probably the only ones worthy of consideration at the present day: *methodical compression of the tumor; simple, repeated puncture of the sac*, repeated according to the necessities of the case; and *puncture, with the subsequent injection of iodine.*

Methodical compression, by means of adhesive plaster, bandages, appropriate compresses, and the like, certainly in some cases effects nothing but an apparent improvement, inasmuch as it mechanically presses the fluid back into the cavity of the spine; as soon as the compression ceases the tumor again returns to its former volume. At the same time, Heister claims to have attained a favorable result thereby. The carrying out of compression by means of *painting with collodion* is highly recommended by Behrend, and has been repeatedly employed with a good result. He first paints the tumor with a mixture of collodion with castor oil, afterwards with pure collodion, and finally applies a compressory bandage.

Experience teaches that *simple puncture* must generally be often repeated before accomplishing its end. Still, even this method is not without danger, even when carried out with the greatest caution, to say nothing of the fact that it is often without any result. The best way is to evacuate the fluid through a very fine puncture (for instance, to draw it out with a hypodermic syringe), not to empty the sac altogether, and afterwards to apply a light compressory bandage.

A surer result, although, perhaps, attended with somewhat greater risk, is to be attained by *puncture with the subsequent injection of iodine.* This method was first introduced by the French, is probably more employed at the present day than any other, and has been especially lauded in England (Morton,[3]

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[1] Berliner klin. Wochensch. 1871. No. 17.

[2] Annal. univers. Vol. 239. p. 143. 1877.

[3] Brit. Med. Journ. April 6th and June 15th, 1872. Lancet, Dec. 2d, 1876.

Watt,[1] Ellis[2]). Morton, in particular, seems to have practise this method with great results; he recently reports fourteen case of which eleven were cured. Recovery followed in *all* the cas seated in the lumbar region—which constitute, as is well know the great majority of such cases. He first makes a test punctur this is repeated in five days, and then *only half* the fluid prese is withdrawn, whereupon an injection is at once thrown in, co sisting of about four grammes of a solution of iodine in gly rine (pure iodine, 0.60 grammes [ten grains], iodide of potassiu 2 grammes [thirty grains], pure glycerine, 30 grammes [ʒ vijss. Others have used other solutions (tincture of iodine and distill water, equal parts; alcoholic solution of iodine and iodide potassium; and some merely alcohol), and have also modifi the method of the injection in various ways, allowing the inject fluid to flow out again, etc. In all such cases it will be well prevent, so far as possible, the penetration of the injected flu into the spinal canal by compression of the pedicle during t injection. The subsequent treatment will be governed by t signs of irritation that may arise.

The main point in any of these operations will always be t careful and accurate carrying out of the same, and the attenti after-treatment.

In cases that are not suited to operative treatment—very larg tumors, very wide clefts of the spine and orifices of communic tion, very feeble subjects—we must endeavor to protect the tu mor, so far as possible, by means of appropriate bandages, com presses properly hollowed out, and the like.

21. *Rara et curiosa.—Rare and Doubtful Diseases of the Spinal Cord.*

In the foregoing chapters I believe I have presented every thing that can now be regarded as to any degree established i the pathology of the spinal cord, and that may be of importanc to the practical physician. That this does not, by any means, exhaust the entire pathology of the spinal cord, I am myself very well aware.

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[1] Brit. Med. Jour., April 26th, 1873.

[2] Philadelphia Med. Times, No. 114. 1874.

There is another class of cases, in which even very serious alterations may be found in the spinal cord, but which are rare in themselves, and, furthermore, by no means admit of a positive diagnosis; cases which are to be regarded more as curiosities, or, at best, as valuable material towards the construction of a future and broader pathology of the spinal cord. There are likewise cases enough which must, in all probability, be localized in the spinal cord, without our being able, at present, to adduce any positive proof to that effect. Finally, there are those in which severe spinal symptoms are merely a part of the manifestations of general processes of disease (severe intoxications, infections, and the like), and are, therefore, more or less disregarded in the general picture of the disease, and usually also receive no therapeutic attention.

Such and such-like matters will be briefly discussed in this section, while, at the same time, I wish to give special emphasis to the fact that, owing to the essentially practical tendency of this Cyclopædia, we are not justified in entering more minutely upon many questions of the highest scientific interest, which are, nevertheless, still awaiting their solution. I must therefore confine myself to more or less aphoristic notices, and would fain, by this means, give the practitioner some stimulus more accurately to observe, and to publish any cases he may encounter of such and similar rare forms of disease.

a. The Pathological Formation of Cavities in the Spinal Cord.—Syringomyelia.—Hydromyelus acquisitus.

G. Namias, Di una specie d'atrofia della midolla spinale. Gaz. med. ital. Lomb. 1851. Nr. 35. (Syringomyelia.)—*W. Gull*, Case of Progress. Atrophy, etc. Hydromelus. Guy's Hosp. Rep. VIII. 1861.—*Koehler*, Monograph. d. Meningit. spinal. Leipzig, 1861. Beob. 17.—*Lancereaux*, Cas d'hypertrophie de l'épendyme spinale, etc. Gaz. méd. d. Par. 1862. Nr. 31.—*O. Schueppel*, Ueber Hydromyelus. Arch. d. Heilk. VI. S. 289. 1865.—Ein Fall von allgem. Anästhes. Ibid. XV. S. 44. 1874.—*John Grimm*, Ein Fall von progress. Muskelatrophie. Virch. Arch. Bd. 48. S. 445. 1869.—*Leyden*, Klinik der Rückenmarkskrankh. I. S. 199. 1874.—Hydromyelus und Syringomyelie. Virch. Arch. Bd. 68. S. 1.

1876.—*Westphal.* Höhlen- und Geschwulstbildung im R.-M. Arch. f. Psych. Nervenkrankh. V. S. 90. 1874.—*Simon,* Ueber Syringomyelie und Geschwul bildung im R.-M. Ibid. V. S. 121. 1874.

Whereas formerly all central cavities developed in the spin cord were regarded at once as dilatations of the central can and, in part, as congenital faults of development, the convicti has recently been reached, through the careful examination o large number of cases, that there are not only pathological di tations of the central canal itself which have been develop secondarily, as the result of various diseased processes est lished in the centre of the spinal cord, the dilatations themsel not being developed till later in life, but that newly formed ce tral cavities also occur within the spinal cord, which have not ing at all to do with the central canal, but lie within the path logically altered tissue, and have been developed out of the sa by various processes of metamorphosis.

All these cases may be grouped together under the nam already used by Ollivier, of "syringomyelia."

After Hallopeau had shown, to a demonstration, that certa chronic inflammatory processes within the spinal cord, which designated as periependymal myelitis, might lead to secondar dilatation of the central canal, Th. Simon, in particular, in large and exhaustive work, established a correct understandi with regard to a great part of the conditions that had previous been thrown together under the name of hydromyelus. On t other hand, Leyden has again recently introduced some new fe tures into the doctrine of syringomyelia which, it would appea necessitate a modification of Simon's views.

Pathogenesis and Pathological Anatomy of Syringomyelia.

Simon sums up the results of his studies on syringomyelia a follows: that an extensive dilatation of the central canal is tole rably frequent in fœtal life, and is rare during later extrauterin existence; that, on the other hand, most of the cases designate as hydromyelus are either of very doubtful authenticity, or cer tainly represent no dilatation of the central canal itself, but are, rather, newly-formed cavities, which are most fond of developing themselves in the foremost portion of the posterior columns, therefore, *behind* the central canal. It is a question here of processes of softening with complete absorption of the softened (and usually also newly formed) tissue. But undoubtedly genuine dilatations of the central canal also take place, arising secondarily through shrinking of the newly-formed tissue which surrounds the central canal. Generally, however, it is a question of newly-developed cavities, which are especially easily formed through the breaking down of tumors that are rich in blood (gliomata, gliomyxomata, etc.), and which may sometimes even be lined with a coating of cylindrical epithelium. Such cavities may be looked upon as the true central canal only when their topographical position with reference to the other portions of a transverse section of the cord justifies this view.

In addition to the above we may add that such *newly-formed cavities* may be developed in very different ways, and as the result of the most varied pathological processes, among which are the following:

Through *breaking down* and *softening in the centre of neoplasms* (gliomata, gliomyxomata, gliosarcomata, etc., cases of Westphal, Grimm, Simon, and others). Not infrequently there is such a complete breaking down that only remnants of the tumor remain, or even only the connective tissue outline of the same. The same tissue as that of which the tumor was formed can then often still be demonstrated above or below the cavity. These centres of softening arise in part through hemorrhage into the interior of neoplasms (so particularly in the teleangiectatic forms), in part through simple softening and cyst-formation within the same. Sometimes there is a development of multiple cavities; once Simon even found a cavity with an epithelial lining (a shut-off portion of the fœtal central canal?).

Through the *breaking down* and *softening* of *apoplectic foci*, by the same process, therefore, that leads to apoplectic cysts within the brain.

Through more or less extensive *central softening in areas of gray degeneration and chronic myelitis*. To this category belong the formation of canals which Friedreich[1] found in a case of tabes of the cord; the formation of cavities which Charcot and Joffroy[2] found in a case of progressive muscular atrophy; the formation of a cleft which Langhans[3] describes in a case of lepra anæsthetica, and more of the same sort.

Finally, through *complete interruption of the continuity* of the spinal cord at some point, as occurred in the experiments of Naunyn and Eichhorst.[4] These observers found, in dogs in whom they had cut or crushed the spinal cord at some point, during their earliest youth, some weeks later, numerous most noticeable cavities, extending a considerable distance upward from the cicatrix. These cavities had, at first, nothing to do with the central canal, and were considered by these observers to be due to an obstruction of the lymph-passages. Westphal, in his case, found something similar in man, and advocates the possibility of a tumor occasionally also inducing such obstruction.

In addition to the foregoing we then still have the *true dilatations of the central canal*, which may arise secondarily as the result of various processes, probably oftenest through the so-called *periependymal myelitis* of Hallopeau, which has also been fully recognized and confirmed by Simon. Here it is a question of a proliferation of the connective tissue surrounding the central canal and of subsequent shrinking of the same, whereby the canal is passively dilated.

Adhesions of the pia mater to the dura mater, *chronic meningeal processes* at certain definite points, appear, according to Simon, also to have a definite relation to acquired hydromyelus—perhaps by a simple extension of the inflammation from the pia mater to the periependymal tissue, perhaps, also, by means of the fact that shrinking meningitic layers of adventitious tissue, by their pulling, mechanically cause the widening of the canal.

Finally, *obliteration of the central canal* at some point

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[1] Ueber degenerative Atrophie der spinalen Hinterstränge. Virchow's Arch. Vol XXVI. Case IV. 1863.

[2] Deux cas d'atroph. musc. etc. Arch. d. Physiol. II. 1869. Second case.

[3] Virchow's Arch. Vol. LXIV. p. 175. 1875.

[4] Arch. f. experim. Pathol. u. Pharm. II. p. 225. 1874.

(through inflammation, external pressure, and the like) may lead to dilatation of the neighboring portions.

Leyden has again recently sought to re-establish a more intimate relation between syringomyelia and congenital hydromyelus. He considers the two as essentially identical, basing his opinion on the observation of two two-year-old children who were affected with hydrencephalocele, and furthermore showed the development of cavities in the spinal cord. Inasmuch as he does not recognize the significance of the tumor-like masses in the neighborhood of such cavities as being that of true tumors, but regards them as mere hypertrophy of the ependyma and of the neuroglia, he comes to the conclusion that congenital hydromyelus is the usual starting-point of the syringomyelia arising later in life, and that even the abnormal cavities situated in the posterior columns are merely remnants, that is to say, extensions of the central canal, hampered in its development. He considers this view as being supported by the history of the development of the central canal, and thinks such cavities should be regarded as portions of this canal which were early cut off. This view may be the correct one for a certain series of cases, but can hardly claim to hold good for all cases of syringomyelia.

The *pathologico-anatomical condition* varies very much in different cases. Cavities of very variable length are met with; often only of a few millimetres or centimetres in length, but not rarely of a length extending throughout the entire spinal cord. These cavities are almost always more or less centrally located, and it is only on more careful examination that we can determine their precise situation and their relations to the central canal.

The width of these cavities varies much—from one to ten millimetres, from the size of a needle to that of one's little finger. Their form, on transverse section, is roundish, oval, angular, sinuate, etc. Sometimes several cavities are found alongside of one another. Their contents consist of a fluid which is light and clear, or turbid yellowish, brownish, etc.; in many cases no accurate statement is made on this point.

The walls of the cavities may be smooth and firm, often provided with a layer of cylindrical epithelium, or sometimes rough, ragged, uneven, etc. In their vicinity we may, according to circumstances, find a hard, cirrhotic tissue or gray degeneration, or the various new formations spoken of above.

The spinal cord, as a whole, is often materially altered thereby, thickened, misshapen; but frequently, also, nothing unnatural can be noticed about it externally. On transverse section the substance of the spinal cord either appears simply crowded and pushed out of place by the syringomyelia, or more or less extensively destroyed, partly through simple degeneration and atrophy, partly through gray degeneration, neoplasms, extravasations, and the like.

Symptoms of Syringomyelia.

All experience thus far teaches that the development of cavities, as such, produces no sort of symptoms by which it could be recognized during life. In many instances the syringomyelia is only accidentally discovered in the bodies of persons who presented no sort of spinal symptoms during life, and who died of some other disease.

In other cases the most manifold spinal symptoms exist: paresis and paralysis, atrophy of muscles, disturbances of sensibility of various kinds, ataxy, paralysis of the sphincters, etc., in the most varied combinations. These are doubtless exclusively the results of the primary alterations (the neoplasms, chronic myelitis, apoplexy, etc.) with which the formation of cavities has been associated later and as an accidental complication. No special symptom, no peculiarity in the course of the disease betrays this complication.

Hence it appears that we are at present possessed of no means to establish the diagnosis of syringomyelia during life. As a rule we shall not be able to entertain anything more than vague conjectures on the subject.

Consequently there is also no question about prognosis or therapeutics. The only indications for the same are to be found in fundamental disease when it is demonstrated.

b. Saltatory Spasm.

Bamberger, Saltatorischer Reflexkrampf, eine merkwürdige Form von Spinalirritation. Wien. med. Woch. 1859. Nr. 4 u. 5.—*P. Guttmann*, Ein merkwürdiger Fall von sog. saltator. Krämpfen. Berl. klin. Woch. 1867. Nr. 13.—*A. Frey*, Ueber den saltator. Reflexkrampf. Arch. f. Psych. u. Nerv. VI. S. 249. 1875. —*P. Guttmann*, Fall von saltator. Reflexkr. Ibid. VI. S. 578. 1876.—*A. Eulenburg*, Lehrb. d. function. Nervenkrankh. S. 699. 1871.

Under the name of saltatory spasm [" saltatorischer Krampf"] Bamberger, in 1859, first described two remarkable cases in which, as soon as the patients placed their feet upon the floor, such a lively clonic twitching of the lower extremities set in that the patients were thrown into the air over and over again, innumerable times—in other words, they fell into a rapid and vehement, involuntary act of hopping and jumping. Afterwards P. Guttmann reported two similiar cases from Griesinger's policlinic, and recently A. Frey, in communicating a valuable case from Kussmaul's clinic, has worked up the question in detail and presented us with a number of suggestive points.

The form of disease under consideration is, on the whole, exceedingly rare. Inasmuch as, according to the unanimous view of all authors, which I also share, it evidently represents a functional disturbance *of the spine*, I consider it justifiable briefly to discuss it at this time.

For the information of the reader the five cases thus far observed will be narrated in outline.

1. Observation of Bamberger (Case 1). A man nineteen years of age had been taken sick with acute pneumonia; during convalescence from the same, severe spasms, of a remarkable character, occurred. *At the moment when the patient's feet touched the floor a tetanic rigidity of his legs took place, accompanied by the most severe concussions, so that the patient was thrown into the air; this was repeated with great rapidity as long as the patient was maintained in an upright position.* At the same time his face wasvery red, his heart's action much accelerated; he had no pain.

In a lying or sitting posture there was not the slightest anomaly to be recognized in the patient. The *sensibility and motility of the legs* then appeared *entirely normal.* But when his feet were pressed against the foot of the bed similar convulsions took place; the spasm could also be originated by *tickling the soles of* *his feet*, and then it sometimes extended to the arm. No atrophy; no pain alon the vertebræ.

In the further course of the case, while the reflex excitability of the lower e tremities still increased, twitchings of the face also set in, as well as increas heart's action, dyspnœa, oscillating movements of the iris, etc.

The administration of morphine seemed to have a decidedly favorable effect the spasm, and in the course of a few weeks recovery took place.

2. Observation of Bamberger (Case 2). *A hysterical woman,* thirty years of a suffering from chlorosis and cardialgia, and complaining of a great general se of weariness. One day when she got up, the same spasms were noticed in her as the first case. *As soon as her feet touched the floor she was at once tossed into th and this was repeated as long as she maintained the upright posture.* Her face was the same time distorted into an anxious expression and the heart's action was tumultuous. The spasm also extended as far up as her head; after the attac certain rigidity remained, accompanied by slight twitchings, for a quarter of hour after the patient was put to bed.

Motility and sensibility seemed normal. Tickling the soles of the feet call forth no reflex action. On pressing the feet against the foot of the bed twitchin occurred only in the upper half of the body. Nothing else particularly abnorm was recognized.

Subsequently excitability was still further increased, to sink again soon a then to remain stationary. At a later period all sorts of hysterical forms of spa were developed. The saltatory spasm did not disappear until after quite a while

3. Observation of Guttmann. A man forty-six years of age. Sixteen ye before, he had fever with *congestion of the head* and *hallucinations.* Afterwards had had frequent repetitions of this congestion, with shooting pains in his limb Three years ago he had the same pains again, followed by trembling in the limb first in the left foot on walking, then also in the right; at that time, already, he h saltatory spasm, which was sometimes stronger, sometimes weaker, but on the who gradually grew worse. Now there is *very well-marked bounding as soon as the patie touches the floor* unless he has a strong support for his arms. *He is flung into the a with great force and rapidity;* his head and trunk are, at the same time, bent bac ward, thereby putting him in danger of falling upon his back. He also suffe severe pain at such times, with a sense of oppression. The spasms are especial severe in the morning, towards evening they often disappear altogether. Psychic disturbances especially favor their occurrence. The attacks are generally precede by a sort of aura in the form of a painful drawing sensation in the legs and bac as far up as the back of the head. He complains of pain along the spine, als sometimes of paræsthesia.

Objectively, *sensibility* seems to be *normal.* *The spinous processes of several ve tebræ are tender on pressure,* and severe pressure on these is sometimes capable o originating a rudimentary attack. *Cutaneous irritation of the soles of the feet without effect.* The spasms only occur at certain times; during the intervals th patient can walk about, stamp on the floor, etc., without inducing an attack.

All therapeutic interference proved unavailing. The patient kept his trouble unchanged (with the aura, and influenced by psychical excitement) until his death, which followed five years later from typhoid fever.

4. Observation of Frey. A man seventy years of age, who had been very much reduced by previous diseases, suffered for two months from rheumatic pains in both legs and in the left arm, which have grown stiff and weak. At a later period *trembling* set in when motions were executed, first in the right leg, then in the left arm. The trembling assumed a more and more convulsive character, and after fourteen days *fully developed spasms* were present. On passive and active muscular movement, severe clonic spasms, starting from the left arm, which is in a state of moderately flexed contracture, show themselves in that arm, throwing the entire extremity upward with great violence about three hundred times in a minute; then they pass over to the right arm and enact just the same scene there. If the left arm is held up still longer, the spasms now pass to the right leg, and finally to the left leg. After a few minutes the intensity of the spasms abates, and they disappear again gradually in the reverse order from that in which they arose. All attempts to produce the spasms by purely *cutaneous irritation* were *unavailing*.

Quite the same state of things existed *in the right leg*. This showed distinct muscular tension; on the attempt to make active or passive movements, severe clonic twitchings (about 300 times in a minute) set up in this leg, which at once passed to the left leg, then to the left arm, and finally to the right arm, gradually disappearing again in the reverse order. Here, too, the spasm could not be called forth by irritation of the skin alone, nor by pressure upon the sole, as long as the foot was not moved.

If the patient attempts to stand on the floor, *as soon as the soles of his feet touch it he is thrown into the air with great force by clonic spasm of the muscles of the calf*, which at first occurs in both calves at once and afterwards becomes alternating, so that *a very curious tripping step, of extreme rapidity, results.* At the same time there is intense pain in the legs. As soon as the patient sits down, the spasms cease.

No disturbances of sensibility. Electrical excitability normal. *Moderate atrophy of the muscles.*

A singular fact was observed, viz.: that the most violent spasms could be quickly quieted by pressure upon the bellies of the muscles of the left arm and forearm in the neighborhood of the elbow (control of reflex action). The same thing was true of the right leg through pressure on the quadriceps femoris a little above the knee.

The malady underwent no improvement under the use of various remedies.

5. Observation of Guttmann: A soldier, nineteen years old, was taken sick with dizziness, headache, great sensitiveness of the spinal column and cramps in his feet. The manifestations quickly increased. At the end of five weeks, *as soon as the patient is set up alone on the floor he is at once thrown into the air, and this is repeated with increasing frequency and violence of the movements until, after a few minutes, the patient threatens to sink to the ground exhausted.* At the same time he has pain in the lower extremities, palpitation of the heart, dyspnœa and a red face. The clonic

spasms are confined to the muscles of the leg and thigh; all the remaining muscles of the body are free. In the *sitting posture*, when the soles of the feet touch the ground, spasms also set in, but more feebly, in the form of *a convulsive tremor of the feet. If the sole of one* foot is tapped with the finger, as in percussion, the clonic twitchings are at once set up; on strong percussion, they are developed even in both legs. The same thing occurs on pressing the feet against the foot of the bed. The twitchings cannot be aroused through any other portion of the body.

No disturbances of sensibility and *none of mobility* in the lower extremities. The spinal column was painful on pressure in three places.

In the course of a year, gradual improvement set in.

These are the only cases thus far known in literature, in which the symptom of "saltatory spasm" has so far stood in the foreground as to be deemed worthy of special notice. Similar conditions of enormously increased reflex excitability are alluded to, here and there, in the pathology of the spinal cord, and yet only in cases that presented much more severe and complicated diseases of the spinal cord. From more recent literature I mention, for instance, the case of Mosengeil,[1] in which, after a traumatic injury of the spinal column, a very high degree of increase of reflex excitability was developed, together with simultaneous paralysis. A recently published case of Chouppe's[2] no doubt also belongs in this category, even though it is not entirely identical with "saltatory spasm." The case is that of a man forty years of age who has been diseased for ten years without change. When he begins to walk nothing abnormal can be noticed, but when he has walked thirty to forty yards the extensors of his legs and thighs contract powerfully and suddenly so that the patient makes a leap that raises him some eight or ten inches from the ground. Then the muscles remain contracted, the limbs are cold, hard, and painful. This condition lasts about a minute. If the patient goes on walking this attack either recurs again soon or he may walk great distances without noticing anything more.

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[1] Berl. klin. Wochenschrift. 1875. No. 43. p. 588.

[2] Contracture des membres inférieures provoquée par la marche. Gaz. méd. de Paris. 1877. No. 11. p. 138. Soc. d. Biolog.

The foregoing five cases have this characteristic in common, that in all *there was a very extraordinary increase of reflex excitability, but only in certain nerve-tracts;* that, as a result of the same, *on standing up, or on touching the sole of the foot to the floor, a singular spasm occurs, which has been very appropriately designated as "saltatory;"* and finally, that *this increased reflex excitability appeared as an isolated fact,* without any other or with but insignificant spinal symptoms; that, in particular, there was no serious paralysis.

An elevation of reflex excitability, even to a very astonishing degree, is by no means a rare manifestation in the pathology of the spinal cord; this can be demonstrated in every instance of myelitis from compression, in every high grade of spastic spinal paralysis, and several other forms of spinal disease. Here, however, it is generally associated with paralysis and other severe spinal symptoms. On the other hand, the almost isolated and quite preponderating occurrence of increased reflex excitability is certainly very rare, and proves that the reflex apparatus as well as the tracts that control reflex action are certainly but rarely diseased alone. But it likewise appears, from the above cases of saltatory spasm, that such isolated or well-nigh isolated disease may yet occur.

The method of action of saltatory spasm has something very peculiar about it; this being thrown upwards as soon as one's feet touch the floor, this severe spasm of hopping, which puts the whole body in motion with great rapidity, is extremely characteristic; it constitutes the most prominent symptom and belongs to *all* cases of the disease, although in some instances various other spasmodic manifestations are also reported.

With regard to the true essence and the starting-point of this peculiar cramp we are not yet altogether in the clear, in spite of the careful observation of cases, and in spite of the peculiarly accurate examinations of Frey. It is a pity that, at that time, the reflex excitability of tendons was unknown. Although Guttmann believes that no relation can be established between this and saltatory spasm, yet I cannot rid myself of the idea that the tendons are the very parts that must decidedly have something to do with these spasms. At least in Frey's case it was positively demonstrated that the reflex action did not originate in the ski Frey regards it as due to the tension and stretching of the m cles, but it is a question whether the tendons were not at t same time also stretched and irritated. The fact that in seve cases pressing the feet against the foot of the bed, and that Guttmann's case tapping on the sole of the foot (*sic!*) arous the reflex spasm, speaks strongly in favor of my opinion. T well-known dorsal clonic spasm of the feet in its higher grad in which both legs fall into a lively convulsive trembling (*tre dation épileptoide*) must, in my opinion, bear a very great semblance to saltatory spasm, and I can readily imagine that t patients who show this phenomenon would also develop salt tory spasm if it were possible to set them on their feet and ke them standing; but they are generally the victims of seve paraplegia. These considerations at all events inspire the wi in future cases more accurately to test the question of wheth saltatory spasm does not bear a certain relation to the refl excitability of tendons—whether it does not represent an increa —certainly to a very high degree—of dorsal clonic spasm of t feet. At the same time, Bamberger's first case seems to sho that the reflex action may also originate in the skin; this, ho ever, is in part also true of dorsal clonic spasm, and may explained in other ways.

There cannot be the slightest doubt that the phenomenon really nothing else than a reflex spasm; this appears, to a de onstration, from all observations, and quite particularly from t case of Frey, in which the subsidence of the reflex manifestatio followed in quite a typical manner. The fact, too, of the chec ing of the reflex action by strong mechanical irritation of t structures lying beneath the skin may also be understood in t same sense.

We have, therefore, in all cases to deal with *a peculiar for of spasm caused by extraordinarily increased reflex excitabili of the spinal cord, and principally localized in certain nerv tracts.*

But does this constitute a distinctive, well-characterized for of spasm, perhaps somewhat similar to tetanus, or is it only o —certainly very striking and remarkable, but yet single—sym tom of disease which may occur in various affections of the spinal cord ?

For the present I should be more disposed to accept the latter alternative. Just as is the case with ataxy, with muscular tension, with partial paralyses of sensation, and many other things, so saltatory spasm might also only be a peculiar symptom belonging to the localization of diseased processes in quite definite portions of the cord. Whether this symptom can be the main and essential one in a certain definite form of disease; whether it will, therefore, be possible, in the future, to gather together cases of this increased reflex excitability under the name of "saltatory spasm," taking only the purest cases and those most free from other manifestations; can only be determined by future investigations. And in these it would be desirable to give special prominence to the more accurate study of reflex action itself and its starting-points, and to settling the question, so far as possible, of all other concomitant manifestations.

The cases at present before us, although they are bound together by the common symptom of "saltatory spasm," can hardly be considered as the same in kind and belonging to a definite form of disease. One case (Bamberger's 2d) is decidedly of hysterical origin; in another (that of Frey) the paresis, the contractures, and the atrophy that were present, speak in favor of the existence of chronic myelitis; in the other three cases (Bamberger's 1st, Guttmann's 1st and 2d) no other spinal functions seemed, to any considerable degree, affected; sensibility and motility were quite normal. In these cases it would at least seem natural to regard them as peculiar forms of disease, whose main symptom was the saltatory spasm. They would constitute the germ of a subsequent more accurate array of cases of this form of disease.

The main causes of this disease would seem to be previous illnesses, debilitating influences, neuropathic disturbances.

The distribution of the spasms differs very much in individual cases; sometimes they are confined to the legs; sometimes they also extend to the back, the neck, and face, even to the organs of circulation and respiration, and to the pupils. In all cases the arms remained free during the actual saltatory spasm, and could be used by the patient for his support. The spasm was almost always accompanied by lively pain in the legs. In one instance the spasm occurred only on standing; in others it could also be called forth by appropriate irritation when lying down Psychical influences could be shown in a number of instance either to favor or to control the spasm.

After giving the above reports of all the known cases, it i not necessary further to delineate the picture of the disease The diagnosis of saltatory spasm will, as a rule, be easy, on account of the extremely characteristic occurrence of the hopping movements as soon as the feet touch the floor.

As regards the *therapeutics* of this manifestation, no great results can yet be shown. In most instances all possible therapeutic measures (warm baths, cold sponging, ice to the back, galvanism, arsenic, iodide and bromide of potassium, etc.) proved entirely ineffective; the trouble remained stationary, or ran its course to a favorable termination, apparently uninfluenced by therapeutic measures. Only in the first case of Bamberger did the administration of morphine seem to exert a decidedly beneficial influence on the spasm.

The treatment of this peculiar condition is therefore still to be discovered. It might be desirable, first of all, to keep our eye on trials of bromide of potassium, calabar bean, ergotine, and atropia (see this Vol., p. 189).

c. Tonic Spasms [Cramps] in Muscles Capable of Voluntary Movement.

Charles Bell, Affections of the Voluntary Nerves. Physiological and Pathological Investigations of the Nervous System. German by Romberg, 1832. p. 367.—*M. Benedict*, Ueber spontane u. reflector. Muskelspann. u. Muskelstarre. Deutsche Klin. 1864. Nr. 30-34.—Elektrotherapie. S. 134 ff. 1868.—*Leyden*, Klinik u. s. w. I. S. 128. 1874.—*J. Thomsen*, Tonische Krämpfe in willkürlich beweglichen Muskeln in Folge von ererbter psychischer Disposition. Arch. f. Psych. u. Nerv. VI. S. 702. 1876.—*A. Seeligmueller*, Ton. Krämpfe in wilk. bewegl. Muskeln (Muskelhypertrophie?). Deutsch. med. Woch. 1876. Nr. 33 u. 34.

After attention had been directed for a long time to certain conditions of *muscular rigidity* which occur in some diseases of the spinal cord, and also in other nervous affections, cases were more recently described by J. Thomsen and Seeligmueller in which such muscular tension and regular tonic spasms, on voluntary motion, formed almost the only demonstrable manifestations of disease. These were cases that probably depended on a congenital anomaly of the nervous system that occurred repeatedly in one and the same family, and were hereditary through several generations—cases which caused the individuals thus attacked great annoyance, and often led to their being falsely judged and unjustly treated.

As these cases are of decided practical importance, and may acquire the most serious significance to the individual—in case of his being subject to military duty, for instance—and as they are, furthermore, probably of spinal origin, they will here be briefly noticed.

We will first, as a sample of this peculiar disturbance, communicate the case very fully investigated and described by Seeligmueller.

Richard Kroitzsch, a recruit, twenty-two years of age, had well-nigh driven his drill-sergeant to despair, because he could not learn to handle his musket with anything like the requisite rapidity and precision, and, furthermore, repeatedly let his piece fall, in the midst of the drill, or even fell to the ground himself, without losing consciousness. The recruit stated that *a certain stiffness and tension in his arms and legs*, especially after long rest of the extremities, *prevented the rapid and precise carrying out of the movements.* An older sister suffers from the same affection.

The patient has been affected with this trouble *from his earliest childhood.* He never could execute rapid movements; was prevented for this reason from becoming a waiter, and was obliged to adopt a sedentary occupation. The stiffness is very great after sitting still for a considerable time, and also on getting up in the morning. It is claimed that *his extremities always grow more supple, and are under better control after prolonged movement;* this also proved to be the case after drilling for a long time. His speech is also somewhat hesitating. His legs and forearms often have the subjective sensation of being asleep and of formication. No pain and never any cramps in the calves of the legs.

Objective examination showed a man of medium stature, with but a slight layer of fat, but with well developed, and in part even athletic, muscles. The domain of the facial nerve and the tongue were normal. Speech good. There was no tenderness on pressure over the spinal column, the nerves or muscles of the extremities. Reflex movement on tickling the soles of the feet increased.

There was *an enormous development of single symmetrical groups of muscles*, thu of the calves of the legs, the quadriceps and trapezius muscles, the glutæi, the bicep muscles, etc. The measurements given showed an unusual size of the individu parts. This massive development is comparatively uniform for the upper and lowe extremities. There is considerable lordosis of the lumbar portion of the spin column. Skin and panniculus normal. Almost all the muscles show an *uneve knobby surface*, which becomes more distinct on motion. The muscles feel *as har as boards*, especially when they are set in action, when they swell up into knott ridges. This is also the case when they are *irritated, mechanically or electricall* On tapping over them, the parts affected at once contract into firm masses, standin out in relief. The same thing takes place on irritation with the faradic curren and it appears, furthermore, that the tetanus thus called forth continues dispropo tionately long (five seconds and more) after the action of the faradic irritation h ceased. The same manifestations were also called forth through a strong galvani current.

In the muscles of the upper arm and shoulder there were distinct *fibrillar con tractions*. The *active movability* of the extremities *did not appear to be materiall disturbed*, during the examination—only the rotation of the shoulder-joint was little slow and hesitating. *Considerable obstacles* were opposed to *passive motion* i most of the joints, particularly the knee-joint. There were no disturbances of co ordination. Climbing stairs was executed with the legs somewhat spread. N muscular tetanus could be called forth by compression of the trunks of the nerve or arteries. The galvanic and faradic excitability of the nerves and muscles prove to be quite normal.

Seeligmueller was induced to report the preceding case by th description which Dr. J. Thomsen had given of this peculiar form of disease. Thomsen himself has suffered from the affectio since his youth, and has demonstrated its existence in his ow children, in his brothers and sisters, and in his ancestors, including numerous cases, and running through not less than fou generations.

The description which he gives, somewhat concisely, of himself and of one of his sons who, by reason of the imperfect judgment formed concerning his case, had to suffer much at the hands of the military surgeons, agrees in all essential points with Seeligmueller's case.

The difficulty begins in earliest youth; Thomsen was able, in part, to recognize it in his children even in the cradle. It consists in a deficiency in the influence of the will on the muscles, which fall into a state of rigidity and stiffness, and even into complete tonic spasm when they attempt to obey the will. This appears the more prominently the more powerfully patients exert their will to overcome the obstacle, the more they are observed, or are the subjects of psychical excitement generally. This condition may become so extreme that patients fall to the ground and tumble around helpless until the spasm abates. On the receipt of sudden severe psychical impressions a sudden painful sensation shoots through all the voluntary muscles. Low temperature, taking cold, and all conditions of disease increase the evil, warmth and movement diminish it. The sphincters are not involved. When the patients once get agoing, and the will has attained its influence over the muscles, then they are as capable of movement as well people.

No other disturbances of any kind are to be observed. Psychically these people are well, although the fear of betraying their condition before people, and the wish to conceal it, rule them and influence their character. There is nothing abnormal to be found in their corporal condition; the muscular system, particularly, may be quite admirable and well developed. In Rostock they excised a piece of muscle from Thomsen's son and found it perfectly normal.

The heredity of the trouble is very remarkable, as is strikingly manifested in Thomsen's family. The great-grandmother of Dr. Thomsen died of puerperal mania, and her two sisters were afterwards psychically diseased. His grandfather was also mentally weak. Of his four children, two had the stiffness to a high degree, the other two (including Thomsen's mother), to but a slight degree. Among the children of this generation, the evil, as well as conditions of psychical weakness, was very wide-spread; of thirteen children borne by the mother of Thomsen, seven suffered from this rigidity. Among the thirty-six descendants of Dr. Thomsen and his brothers and sisters, on the other hand, but six suffer from this complaint. Four of his own five sons have the disease. This is, therefore, a very classical example of "neuropathic encumbrance" ["neuropathischer Belastung"] in a family.

I furthermore find a case in Leyden (l. c.) which plainly belongs here, and some details of which I wish to mention, as it may serve in certain respects to complete the description of Seeligmueller.

A merchant, twenty-eight years of age, whose brother suffers from a similar muscular affection, and whose muscles are developed in quite an athletic manner shows a striking stiffness in all his movements, which he has had from his earliest youth. Individual groups of muscles do not promptly obey the will, and, when a certain movement is intended, come to a stand-still half way in a state of tetanic rigidity. His speech is slow and awkward, as are also the movements of the tongue Some of the motions of the eyes, and of those muscles controlled by the facial nerve, are also sluggish. If the patient clenches his fist powerfully it is impossible for him to extend his fingers again immediately; this is accomplished but slowly, as if he had to overcome a serious obstacle. After these attempts have been repeated a number of times the movements are accomplished more easily. The same manifestations occur in the lower extremities, etc. Aside from this the patient is perfectly well. The malady remained uninfluenced by any treatment.

In Benedict, too, there are some observations in which this appearance of muscular stiffness stood out in a very prominent manner. This is particularly true of observation No. 88, in his "Electrotherapy," while observation No. 86 doubtless represents a somewhat more complicated, but, in certain respects, a similar case.

Charles Bell, too, has undoubtedly seen the affection here discussed, and has described it—even though in quite hasty lines—in that portion of his celebrated work cited above.

I myself very recently observed a case which, at least, gave indications of quite similar manifestations. The patient, twenty-one years of age, came to me on account of symptoms indicating multiple sclerosis in process of development, and stated that from his youth up he had been the butt of his comrades on account of his being so stiff and unhandy. He had always felt a cramp-like stiffness in his limbs whenever he endeavored to use them rapidly, especially if he was being observed by others. This was noticeable in rising from a chair, in going up stairs, etc.; if he took a firm hold of any slender object with his hands, he was unable to let go of it again immediately, etc. This condition was worse in the cold. When once started in walking his movements followed much more easily.

Signs of these conditions are often enough met with in persons with disease of the spinal cord, but then they are always complicated with other and more severe spinal symptoms. This peculiar stiffness and awkwardness of motion, and the fact that many patients walk and move worse when they are being observed, are known to every experienced physician.

On the other hand, the almost isolated occurrence of these disturbances, as it appears in the cases given above, is certainly a rarity, though it might be worthy of the special attention of military surgeons.

Briefly stated, the picture of the disease, as based upon the scanty material at hand, is about as follows:

From their earliest youth—doubtless, as a rule, under the influence of hereditary conveyance—these patients suffer under a peculiar disturbance of movement, which is but rarely developed for the first time in adults. This disturbance of movement consists in a peculiar stiffness and rigidity of the muscles which are intended to be set in action by the will; a stiffness which may increase to a regular tonic cramp, so that movements are entirely hindered, the patients fall to the ground, etc. Just as the voluntary contraction of the muscles is opposed by a strong internal obstacle, so the termination of a contraction that has once set in also follows but slowly, as if overcoming an obstacle; the contractions which were accomplished with difficulty persist long—patients cannot at once let go of articles which they have seized firmly. It is only when, after repeated powerful efforts of the will, the tracts for motor excitation seem, as it were, to have become free again, that movements follow with increasing freedom and ease, and are but little to be distinguished from healthy movements. Psychical influences, sudden mental impressions, the effort of paying attention, the presence of strangers, cold, etc., act unfavorably on the condition; warmth, moderate movement, a good state of mind, a high degree of self-confidence, relieve it. Aside from this these patients are well; their bodily and mental functions are accomplished normally; their muscular system may be admirably developed and even capable of accomplishing a large amount of labor. Sometimes (always ?) tension of muscles takes place on making passive movement, and on mechanical and electrical irritation of the muscles strikingly powerful tonic contractions of the same set in. With all this such patients are unfit for many of the duties of daily life and for the exercise of various callings, and above all are unqualified for military duty.

We will not here enter into far-reaching speculations with regard to the *essence* of this remarkable form of disease. Thomsen thinks that it depends on a disturbance falling principally within the domain of psychical influences. Seeligmueller does not share this opinion. Whereas he originally thought it to be a peculiar form of muscular hypertrophy, the doctrine of a congenital or inherited affection of the lateral columns of the cord afterwards appeared to him most probable. I must confess that I have thus far been unable to form any definite idea of the seat and the variety of affection lying at the foundation of this difficulty, although, owing to the occurrence of analogous symptoms in so many diseases of the spinal cord, I should first of all think of a spinal affection. The question can only be decided by further observation.

There is also wide play left here for *therapeutic endeavors*, which, in the cases thus far reported, seem to have been entirely without results.

d. Intermittent Spinal Paralysis.

Macario, Gaz. méd. de Paris. 1857. Nr. 6.—*Romberg*, Lehrbuch d. Nervenkrankh. I. p. 752.—*Hartwig*, Ueber einen Fall von intermittirender Paralysis spinalis. Diss. Halle. 1874.

The occurrence of purely intermittent attacks of paraplegia and paralysis, which, according to all appearance, are of spinal origin, and which make a most striking impression as contrasted with the usual stability of spinal paralysis of similar severity, is undoubtedly one of the rarest and most remarkable forms of manifestation of malarial infection.

Literature contains but very few instances of this form of spinal paralysis.

I find one case of Macario's cited in Hertz,[1] in which a woman, two days after her confinement, was attacked with formication in her feet without any known cause, which extended over the trunk to the upper extremities. The extremities became paralyzed and anæsthetic, and the tongue, too, was paralyzed. These manifestations were repeated in a quotidian type three times, and were cured by quinine.

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[1] This Cyclopædia, Vol. II., p. 601, foot-note.

Romberg describes quite a similar case.

A woman, sixty-four years of age, after being quite well the day before, was suddenly attacked with paralysis of the lower extremities and the sphincters. Sensibility was unchanged, consciousness clear, the temperature cool, pulse 80, small and empty, no pain in the spinal cord. The next day there was an astonishing change in the condition. The patient can walk again, and void urine voluntarily, and only complains of weakness in the legs. The next morning there was paraplegia again, which had set in at the same hour as two days before. A third paroxysm was awaited, which also set in at the appointed time, although without paralysis of the sphincters. Quinine effected a rapid cure.

In a recent dissertation at Halle, Hartwig has reported in detail an observation belonging under this head.

This observation is in relation to a vigorous laborer, twenty-three years of age, who had suffered from tertian intermittent for a few weeks five years before, but since that time had remained perfectly well and strong. In November of 1873, he first noticed weariness in the legs, which gradually increased, and the arms, too, were attacked. On the third day he was obliged to take to his bed, and the night following he was completely paralyzed—his legs, trunk, arms, and even the movements of his head were paralyzed, but not the muscles of the face; speaking, breathing, and swallowing were somewhat hindered; there was no paralysis of the sphincters, sensibility was intact, the head entirely free, no pain. The secretion of sweat was excessive. After this condition had lasted for twenty-four hours it let up, and in half an hour, generally with an increased secretion of sweat, all the muscles again became movable.

During the succeeding twenty-four hours the patient remained free from any sign of paralysis, merely complaining of weariness and heaviness of the limbs. Then the attack of paralysis recurred again, in the same way as at first, and then there followed regular successive free intervals and attacks, both of about twenty-four hours' duration. Gradually the time occupied by the attack extended to forty hours, the interval being much shorter. Under the use of arsenic the intervals also extended over a period of about forty hours. On first using quinine the attacks remained altogether absent for four days. After hypodermic injections of strychnia the tertian type of the attacks was re-established. Between two days, on which the patient is completely paralyzed on both sides, he has a day of tolerably free mobility of all his members. The sphincter ani was likewise affected a few times; the general condition of the patient had not materially suffered.

At the end of March, 1874, the following condition was found to exist during an attack: The patient lay there completely paraplegic; only the muscles of his face acted normally; the flexors of the hands and feet show a minimum amount of motion. The pupils reacted well; the special senses were normal. The head could not be moved; on inspiration, it was principally the diaphragm that was active; expiratory efforts were reduced to a minimum. Speech and swallowing were somewhat difficult. Respiration 20, pulse 72, temperature 99.5°; sensibility of the skin and muscles normal. Reflex action is entirely wanting; the electrical excitability of the muscles is almost entirely extinguished (during the intervals it is only diminished). No pain, only formication and a feeling of numbness in the parts affected. Evacuations voluntary.

In the course of the succeeding months greater and lesser irregularities occurred in the course of the disease. The use of quinine sometimes prevented the attacks for a number of days, but then they returned again in spite of its continued use. On the whole, however, the disease retained its intermittent or strongly remittent type, and gradually a certain degree of cachexia began to show itself, with marked emaciation of the muscles, etc. After the trouble had lasted for seven and a half months no radical improvement could, as yet, be demonstrated, and nothing is stated with regard to the final termination of the case.

These three very remarkable cases have the following features in common: that they all present a rapidly developed paraplegia, advancing to complete motor paralysis, with or without anæsthesia and paralysis of the sphincters; that this paraplegia disappears again in the course of a few hours, sometimes with the appearance of a critical sweat, to give place to a complete or well-nigh complete intermission; that this process is then repeated, in a more or less regular manner, in the quotidian, tertian, or quartan type; and that the entire affection is either promptly cured by quinine, or at least favorably influenced thereby.

It is in the highest degree probable that we here have to deal with a malarial infection; the intermittency of the paroxysms, their termination in a sweat, and the efficacy of quinine, all argue strongly in favor of this intermittent paraplegia being nothing else than a masked intermittent. To be sure, this cannot, as yet, be considered as rigidly proven.

It may probably be even more positively asserted that the seat and starting-point of this disturbance is to be found in the spinal cord. The entire character and grouping of the symptoms, the limitation of the paralysis to purely spinal nerves, the entire immunity of the brain, the great resemblance to other general paralyses of notoriously spinal origin, speak with such positiveness in favor of this belief that no reasonable doubt on the subject can be raised.

But it is, unquestionably, a matter of the greatest difficulty to determine what actually takes place within the spinal cord in this malarial paraplegia,—whether any, and, if so, what anatomical changes take place therein during the attack. Hartwig assumes that it is a question of transitory hyperæmias and serous transudations in the substance of the spinal cord; a supposition which appears to me untenable, from the mere fact that the disturbance is always confined to the motor portions of the cord, leaving the sensitive portions entirely free. It is probably safer to say that the essential conditions of malarial spinal paraplegia are at present unknown to us, and that we can only hope for light on this subject from the future.

The *diagnosis* of "intermittent spinal paralysis" is very easily arrived at from the picture of the disease. And when the diagnosis is clear the *treatment* is plain. The treatment applicable to intermittent fever in all its forms—quinine, arsenic, removal from a malarial region, etc.—must be undertaken with energy. If this remains without results, then the therapeutic measures laid down in various portions of this work for acute and chronic spinal paralysis will claim consideration.

e. Toxic Spinal Paralysis.

Raoul Leroy d'Etiolles, Des paralysies des membres inférieures. II. p. 1–75. Paris, 1857.—*Jaccoud*, Les paraplégies et l'ataxie du mouv. pp. 321–334. Paris, 1864. *Leyden*, Klinik. II. 1. Abth. pp. 280–297. 1875.

In connection with the foregoing cases of infectious spinal paralysis, it may, perhaps, be appropriate to give some particulars with regard to similar instances of paralysis called forth by known and actual poisons.

It is claimed that such paralysis (in various forms, as paraplegia, as paralysis of single groups of muscles or extremities, as general paralysis, etc.) has resulted from intoxication with arsenic, phosphorus, lead, quicksilver; furthermore, as the result of poisoning with carbonic oxide, sulphide of carbon, tobacco, camphor, ergot, lathyrus sativus, alcohol, absinthe, mushrooms, balsam of copaiba, etc., entirely apart from the severe acute paralysis of the nervous system, such as is called forth by opium, belladonna, strychnine, etc.

All these poisons are distinguished for their powerful though varied influence on the nervous system; they cause lasting paralysis, as a rule, only when their action is slow and repeated, more rarely when it is quite acute. They cause the most varied forms of paralysis and paraplegia, from simple weakness and paresis to complete paralysis, sometimes acute, sometimes more chronic, with or without muscular atrophy, with or without disturbances of sensibility. In many cases, however, the causal connection between these and the antecedent intoxication is by no means placed beyond all doubt.

Furthermore, we know almost nothing with regard to the changes that may exist in the nervous system (especially in the spinal cord) in such paralyses. We have virtually no anatomical reports on this subject that carry any weight with them; the localization in the spinal cord cannot even be asserted, with any degree of positiveness, on the ground of the clinical symptoms. Great uncertainty still reigns even with regard to the most frequent and best known of these forms of paralysis, viz., lead-palsy. What is to be said and conjectured on this subject has already been given in other portions of this work.

The pathology of the spinal cord has, therefore, not as yet experienced any advancement through the study of toxic paralysis, and there is all the less reason why we should here enter upon these matters in detail, because this would carry us into the domain of toxicology, this being the field where, for the present, the scientific working up of these questions will have to take place. The reader who is more particularly interested in this direction will find that, aside from the hand-books on Toxicology, the works above cited contain more detailed accounts of these matters, even though the results may still be unsatisfactory.

f. Paraplegia dependent on Idea.

J. Russell Reynolds, Remarks on Paralysis and other Disorders of Motion and Sensation dependent on Idea. British Med. Journ. 1869. Nov. 6.

In a very readable essay, and one of importance to the practitioner, Russell Reynolds some time ago pointed out the fact that severe disturbances of the nervous system (paralysis, cramp, pain) may depend upon diseased action of the mind, and upon imagination; that such disturbances, under the garb of affections of the brain or spinal cord, may long bid defiance to all treatment, and only disappear after the removal of the erroneous ideas. Such conditions may occur quite independently of psychoses, of hysteria, hypochondriasis or simulation; they are often associated with general debility, and even with actual, well-defined diseases of the nervous system, and, if a correct diagnosis is made, may easily be removed by appropriate treatment. Everybody knows the powerful influence which imagination and lively mental operations have upon pathological manifestations in the nervous system, and we are not surprised if these influences make themselves felt in the domain of sensation and feeling; we are less accustomed to the idea of such psychical impressions manifesting themselves in the form of severe motor disturbances also, although there is no reason, *a priori*, why this should be impossible.

Taught by our own experience that such conditions actually do occur, and perhaps occur more frequently than one would think, we consider that it would not be amiss to give a brief representation of Russell Reynolds' views, so far as they refer to disturbances in the sphere of the spinal functions. The reader will agree with us that the easiest way to familiarize him with the subject is by the brief narration of the most significant one of Russell Reynolds' cases.

A young lady who had seen better days was admitted into the hospital as paraplegic. She had become so gradually, and was much emaciated. She had been unable to stand for two or three months, and was now bed-fast. Her expression of countenance was anxious, mixed with a glimmer of hope; she expected to be cured as the result of entering the hospital,—a step upon which she had resolved with difficulty.

Her paralysis was almost complete; she could move the toes somewhat, and raise the heel a little from the bed. But the sphincters were normal, and there was no local atrophy. Sensibility was intact; electro-muscular sensibility and contractility normal; reflex action hard to accomplish; no conditions of cramp; no pain, even on movement of the legs or on pressure over the spinal column. No hysteria; no traumatic injury; no cachexia demonstrable.

Her previous history is as follows: Her father, who is her only relative, was brought from affluence to poverty, a year and a half ago, through commercial disasters. He bore it bravely, as did also his daughter; he set to work again, and she assumed duties and responsibilities which she had never known before. At first all went well, but suddenly the father became paralytic, and his daughter nursed him with tender self-sacrifice. But they soon found themselves in actual want. The daughter worked as daily governess, was obliged, as a matter of economy, to walk a great deal, and walked fast, so as to gain time for her father. Thus she worked for many a weary week, the thought of paralysis always before her, her brain overburdened with thought and anxiety, her limbs weary with walking, her spirits hardly equal to the constant effort to appear cheerful. Her legs often hurt her, and she thought, with shuddering, what if she should grow lame like her father! This thought acquired an increasing power over her; she was gradually obliged to give up walking, then to stay at home, then in her room, and finally in bed. Her legs grew more inactive from day to day until complete paralysis was present.

She was told, and all those about her were told, in confidence, that she would soon be able to walk perfectly well. A light tonic was administered; her legs were faradized, though more for the sake of the psychical impression; her legs and back were rubbed and she was obliged to walk up and down the hall for five minutes every four hours supported by two nurses. The day after the beginning of this treatment she could walk with but little support. After four or five days she could walk quite well, and at the end of two weeks she was as strong and capable as ever before in her life.

Not less interesting are the other examples which Russell Reynolds gives. I have likewise met with quite similar things in my own practice, even if not so well marked in degree, and have repeatedly seen improvement set in through appropriate psychical impressions made upon the patient.

It is, of course, by no means always easy to recognize such conditions aright and to separate them from real diseases with an anatomical basis. Such conditions are by no means always associated with hypochondriasis or hysteria, or mental disturbances, although this may occur: still less are we to suppose that such patients are indulging in malicious simulation; they are themselves firmly convinced of the reality of their symptoms. A certain general debility not rarely accompanies such conditions, and all possible debilitating influences—diseases, cares, anxieties, overexertion, etc.—may be followed by the same. But it generally requires, in addition, that the attention be habitually directed to certain portions of the body or to certain forms of disease in order to call forth such "imaginary" disease. On this ground Russell Reynolds believes that a part of the neuroses caused by railroad accidents may be classed under this head, wherein the attention of the victim is influenced in the most unfortunate manner by the stories of friends, by the inquiries of his physician, the talk of his attorney, and the sober face of the company's physician.

The *diagnosis* of such forms of disease is based on the following circumstances: on the method of development of the symptoms, and especially on the demonstration of some ruling idea of the mind; on a judgment with regard to the symptoms present, and especially on the demonstration of such symptoms as cannot be reconciled with the theory of an organic lesion. In this respect Russell Reynolds calls attention to the following points: that the patient cannot lift his heel from the bed nor draw it up, while he can raise himself up or lie down without any help, or turn from one side to the other; that in apparently complete motor paralysis there are no signs of paralysis of sensation, no trophic disturbances and no diminution in the electrical excitability; that sometimes there is complete inability to stand on one's legs, whereas the legs can be moved in every possible direction. Finally, the diagnosis is also said to be aided by the failure of ordinary treatment and the success of treatment directed to the removal of the false idea. That it will not always be possible to make a positive diagnosis on the ground of these signs, is a fact which will be admitted by every one familiar with the pathology of the spinal cord.

In the way of treatment Russell Reynolds gives the following recommendations: Let the case be taken hold of earnestly; excite the hope of recovery, if a definite plan of treatment is followed out, and let this be done not only by the physician but by those around the patient—hence the importance of hospital treatment; compel the patient to walk at certain definite times, supported on both sides; use faradization to the muscles, friction and passive movements of the limbs; apply dietetic and medicinal treatment such as the general condition of the patient requires.

So far as we know, these matters, which are certainly not without practical importance, have thus far not received the attention which they unquestionably deserve. It would be interesting to receive communications from others regarding similar manifestations; perhaps it would be possible after a while to gain somewhat more insight into the pathogenesis of these processes, which thus far almost elude all scientific explanation.

DISEASES

OF THE

MEDULLA OBLONGATA.

ERB.

I. Anatomical Introduction.

The chief works to be consulted are: *B. Stilling*, Untersuch. üb. d. Textur u. Functionen der Medulla oblong. Erlangen. 1843.—Untersuch. über den Bau des Hirnknotens oder der Varol'schen Brücke. Jena. 1846.—*Schroeder v. d. Kolk*, Bau u. Function der Medulla spin. u. oblongata. Braunschweig. 1859.—*Deiters*, Unters. üb. Gehirn u. Rückenmark des Menschen u. der Säugethiere. 1865.—*Stieda*, Ueber den Ursprung der spinalartigen Hirnnerven. Dorpat. med. Zeitschr. II. 1873.—*Meynert*, Stricker's Handbook. Vol. 2.—Skizze des menschlichen Grosshirnstammes, etc. Arch. f. Psych. und Nerv. IV. 387–431. 1874.—*Henle*, Handbuch der Nervenlehre. 1871.—*Huguenin*, All. Pathologie der Krankheiten des Nervensystems. I. Anatom. Einleitung. 1873.—*Flechsig*, Die Leitungsbahnen im Gehirn u. Rückenmark des Menschen. Leipzig. 1876. —Ueber Systemerkrankung im Rückenmark. Arch. d. Heilk. XVIII. 1877.—*Farabeuf*, in Diction. encyclo. des sciences médic. 2 Sér. tome VIII. p. 299. 1874.—*W. Krause*, Handbuch d. menschl. Anatomie. I. 1876.—*Duret*, Sur la distribution des artères nourricières du bulbe rhachidien. Arch. de Phy. norm. et pathol. 1873. p. 97.

Many points, and these perhaps the most important, in the *anatomy of the medulla oblongata*, are still unsettled. Indeed, the relations here are, more than elsewhere, difficult to disentangle. The connections between the different structures of the medulla oblongata and those, first, of the spinal cord below, and second, of the central nervous system above, are so extremely complicated, numerous, and entangled, that even the most careful and thorough anatomical investigation can scarcely make them understood. This is quite sufficient explanation for the fact that the views of the best observers on the finer structure of the medulla oblongata are still at great variance with one another on several points.

The space in this handbook does not allow of an accurate and exhaustive description of the outward form and internal structure of the medulla oblongata. Yet we can scarcely avoid giving a short sketch of the principal points, in order to refresh the practitioner's memory on anatomical relations, to put him *au courant* with the latest investigations, and acquaint him somewhat with the present nomenclature, and thus, we hope, facilitate his comprehension of the morbid anatomical details in the following pages.

The best way of attaining this object will be to begin with the structures of the spinal cord, with which we are already acquainted, and follow these into and through the medulla oblongata, and after this to give a short description of the other parts which are peculiar to the medulla. To suit the practitioner's interests, we shall endeavor, first of all, to give a clear and correct idea of the topography of the parts, leaving aside the details of the course of the fibres and their manifold connections, partly because these are wholly uncertain, and in many instances in great part unknown.

Even the external boundaries of the medulla are still a matter of dispute among anatomists, though every one agrees to bound it *inferiorly* with the roots of the first cervical pair of nerves.

The superior border, however, is undecided, some considering it as formed by the inferior border of the pons (which would correspond on the posterior surface to the upper striæ acusticæ); but most writers include the whole floor of the fourth ventricle, *i. e.*, the whole rhomboid sinus; these latter boundaries, too, are better suited for clinical purposes. We should then take for the upper boundary the processus cerebelli ad corpora quadr. (which form the lateral walls of the fourth ventricle) and the entrance of the Sylvian aqueduct; thus, we may regard the pons as a cerebral structure, which bounds the medulla oblongata on one side, and then we need concern ourselves no more with its structure or pathology in this article.

From a superficial examination of the exterior, one would be led to think that the columns of the spinal cord run without interruption into the structures of the medulla oblongata; but closer observation proves the error of this view, and shows that the most manifold transpositions take place, which contribute to bring new and important structures to the surface, and produce the characteristic appearance of the medulla.

If we follow up the anterior spinal columns, they appear to ascend directly and run into the pons. But, on drawing apart

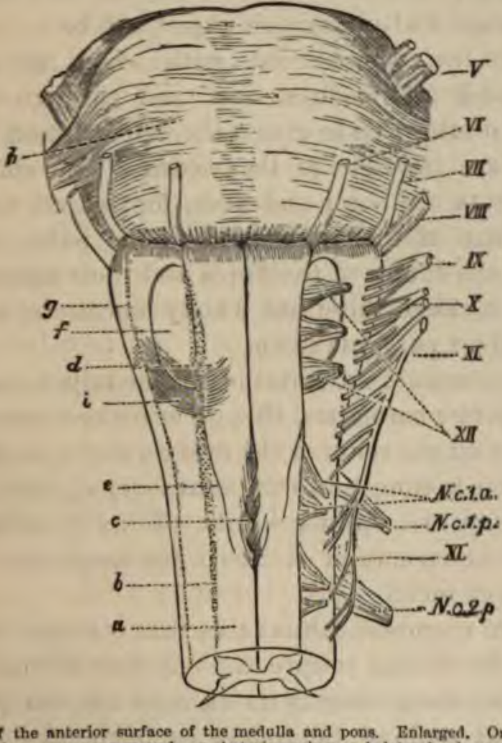

FIG. 20.—View of the anterior surface of the medulla and pons. Enlarged. On the right side of the medulla the roots of the nerves are removed. *a*, Anterior column; *b*, lateral column; *c*, decussation of the pyramids; *d*, pyramids; *e*, groove between anterior column and pyramid; *f*, olivary body; *g*, corpus restiforme; *h*, pons; *i*, fibræ arciformes; *V–XII*, roots of the fifth to twelfth cerebral nerves; *Nc1a*, anterior roots of the first cervical nerves; *Nc1p*, posterior roots.

the lips of the anterior median fissure, we perceive that, at the level of the first cervical nerves, it is obstructed for a distance of from six to seven mm. by coarse bundles of fibres, which pass from one side to the other, *decussating* in fact, and then continuing their course on the opposite side, along the anterior fissure. Here we see a crossing of fasciculi, which come to the surface from the interior, and then usurp the position of the anterior columns in their course upwards. This is the so-called *decussation of the pyramids* (Fig. 20, *c*), and the columns which go from this to the pons, and take the place of the anterior columns, are called the *pyramids* (Fig. 20, *d*). They issue from between the anterior columns (Fig. 20, *a*), which latter retreat and disappear into the interior of the medulla. This arrangement is often rendered evident by a distinct groove (Fig. 20, *e*) running obliquely upwards, and forming the boundary between the pyramids and the anterior columns proper.

The *lateral columns* of the spinal cord proceed directly upwards through the medulla, and are now surrounded by the (motor) roots of the hypoglossus (Fig. 20, *XII*) and the (chiefly sensory) roots of the glossopharyngeus (*IX*) and vagus (*X*), which replace the anterior and posterior roots of spinal nerves. In the upper half, almost immediately behind the pons, projects an almond-shaped structure, like a long prominence, and bordered on either side by attenuated stripes of the lateral columns; this is the *olivary body* (Fig. 20, *f*). Its prominence is subject to considerable individual variation. The upper portion of the lateral column is here often called the *olivary column*. A small portion of the lateral column (Flechsig's direct cerebello-lateral-columnar tract), belonging to the periphery of the postero-lateral column, passes in the medulla obliquely backwards, and takes part in the formation of the corpus restiforme, and also of the pedunculi cerebelli.

The *posterior spinal columns* at first continue their parallel course upwards; a little above the superior termination of the decussation, however, they part at rather a sharp angle, and pass into the cerebellum under the form of round columns, much increased in size. These columns are the *pedunculi cerebelli* (Fig. 21, *g*) or *funiculi restiformes*. The increase in size would alone indicate that these are not the mere prolongation of the posterior spinal columns; in fact, new bundles contribute to the formation of the cerebellar peduncles. The components of the posterior columns proper appear to run directly into the pedunculi; the funiculi graciles (Goll's cuneiform columns) (Fig. 21, *b*), forming the internal portion of the pedunculi, at the point of the angle swell to nodules, the *clava* (Fig. 21, *d*), and are usually connected with the cuneiform column of the other side by a slender band of white substance, the *obex* (Fig. 21, *e*).

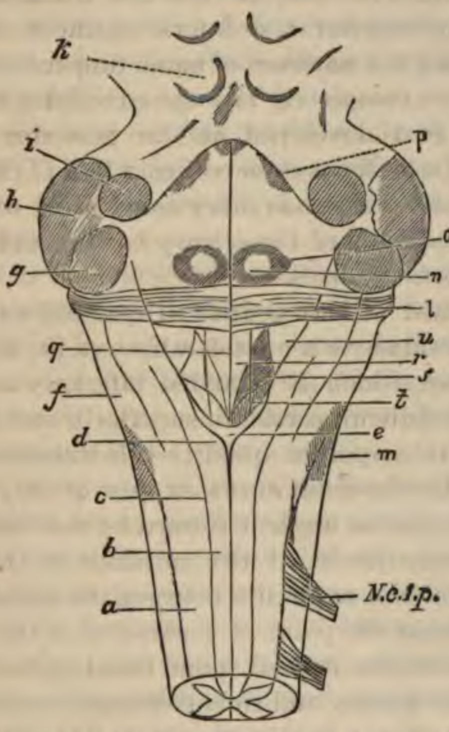

FIG. 21.—View of the posterior surface of the medulla, the roof of the fourth ventricle being removed, to show the rhomboid sinus clearly. The two halves are drawn somewhat apart. Enlarged. *a*, funiculi cuneati, et *b*, graciles; $a + b$ = posterior spinal column; *c*, sulcus intermedius posterior; *d*, clava; *e*, obex; *f*, corpus restiforme; *g*, pedunculus cerebelli $(= a + b + f)$; *h*, crus cerebelli ad pontem; *i*, crus cerebelli ad corpora quadr.; *k*, corpora quadr.; *l*, striæ medullares s. acusticæ; *m*, fibræ arciformes; *n*, eminentia teres (genu nervi facialis); *o*, fovea anterior; *p*, locus cœruleus; *q*, funiculus teres; *r*, nucleus of the hypoglossal; $s + t$, ala cinerea; *s*, nucleus of vagus; *t*, nucleus of accessorius; *u*, nucleus of acusticus; *Nclp*, posterior root of the first cervical nerve.

The *sulcus intermedius posterior* (*c*), which divides the gracilis from the *cuneiform column* proper, continues its course up to the striæ acusticæ. The *cuneiform column* runs upwards alongside the sulcus, increasing considerably in size, as it is reinforced by other fasciculi, which form the lateral half of the pedunculus cerebelli. These fasciculi receive the name, *corpus restiforme* (*f*), and are frequently marked off from the cuneiform column by a distinct groove (dotted line in Fig. 21), so that they have been divided by some (Henle) into median and lateral cuneiform columns; this latter distinction can be more easily detected in a transverse section (Huguenin). The corpus restiforme is partly made up of superficially situated bundles, which have been described under the name of *fibræ arciformes* (Fig. 20, *i*; Fig. 21, *m*). These arise partly from the bottom of the anterior median fissure, between the pyramids, and partly from the lateral fissure, between the pyramids and olivary bodies. They appear to be derived from the anterior columns, and form an arch round the anterior and lateral surfaces of the medulla, sometimes of large, sometimes of small dimensions; finally, they disappear almost completely into the cuneiform columns. They are generally best developed at the posterior angles of the olivary bodies, sometimes even covering these. But the corpora restiformia receive numerous other bundles of considerable size from the neighborhood of the olivary bodies, and probably, too, from the bodies themselves.

The separation of the pedunculi cerebelli exposes the deep-seated gray substance to a considerable extent, thus producing a slight depression, which is bounded inferiorly and laterally by the diverging pedunculi cerebelli, superiorly and laterally by the converging crura corporum quadr.—this lozenge-shaped depression is called the *rhomboid sinus*, or *floor of the fourth ventricle* (Fig. 21). Its superior angle is formed by the union of the crura cerebelli ad corp. quadr. at the entrance to the Aquæductus Sylvii, and its inferior angle lies between the pedunculi cerebelli, behind the obex at the point of separation of the funiculi graciles. Just here too the central spinal canal opens into the posterior longitudinal fissure, and thus communicates with the fourth ventricle. The obtuse angles on either side are formed by the junction of one crus with the peduncle of the corresponding side.

The floor of the fourth ventricle is divided symmetrically by a longitudinal shallow groove. A superficial inspection shows us here a number of grooves, depressions, projections, and gray patches, etc., which are all connected with important structures in the interior, and thus of practical importance to us.

Of these certainly the most striking are the *striæ medullares* (Fig. 21, *l*). In numbers varying from one to twelve, these bundles run horizontally at right angles to the median fissure, from which they emerge, and cross the pedunculi, and, passing round them, join the nervus acusticus. They divide the sinus into a superior large, and an inferior smaller space. Above the striæ, and in the right angle formed by them with the median fissure, lies a small, rounded, white prominence, corresponding to the *genu nervi facialis* and the neighboring *nucleus of the abducens* (Fig. 21, *n*). Laterally and anteriorly to this we find the so-called *fovea anterior* (Fig. 21, *o*), a depression which is generally colored with a dark pigment, and presents the appearance of a contracted scar. At some depth below this lies the *nucleus* proper *of the facial* nerve.

Going from this towards the superior angle of the fourth ventricle and slightly outward, we strike another gray depression, called the *locus cœruleus* (*p*), which borders on the motor nucleus of the trigeminus.

The gently elevated cords which traverse the inferior triangle of the rhomboid sinus, parallel to the median fissure, receive the name of *funiculi teretes* (Fig 21, *q*). Beneath them lies on either side a *nucleus of the hypoglossal* (Fig. 21, *r*), extending longitudinally for some distance. Passing outwards, we are met by a light-gray triangle, with its apex pointed to the striæ, and its base resting on the clavæ. This is called the *ala cinerea* (*s* and *t*). Its anterior portion (*s*) is coextensive with the *nucleus of the vagus*, while the posterior (*t*) contains the upper end of the *nucleus accessorii*, which extends far down into the spinal cord. In the interior of the medulla, somewhat anterior to the ala cinerea, the *nucleus of the glossopharyngeus* comes in contact with that of the vagus. The *posterior median nucleus of the acoustic* forms the anterior and lateral boundary of the vagus nucleus. It comes nearer the surface than the glossopharyngeus, and occupies nearly the whole space between the ala cinerea and pedunculus, up to the upper border of the striæ. Henle has described it under the name of *nucleus acusticus superior*.

The *internal structure* of the medulla oblongata is so extremely complicated that no one has yet been able to throw the slightest light on the general course of the fibres and the connection between its paths of conduction. Though some structures have been recognized as undoubtedly analogous to certain components of the spinal cord (gray nuclei—gray columns, commissures, etc.), still we meet with so many new and problematical forms, the different parts combine in so various and complicated manners, and the fibrils run in such entangled, tortuous and interwoven courses, that we must almost despair of ever following them up accurately. In fact, up to the present the most careful and exact investigations have thrown but a very feeble light upon the subject.

Very little is known with certainty and accepted by all, and the few facts of this nature may be well described as landmarks in an unknown region. With the rest we need not further occupy ourselves, since there is no practical value to be derived from studying all the conflicting views of the hour. Our task in this direction only extends to the indication of those regions about the structure of which next to nothing is known.

The value and significance of successive sections, which are here too the chief method of investigation, can be studied in anatomical text-books. From the nature of the subject we can imagine how imperfect must be the results.

The first material change in the spinal cord—that, indeed, which leads to the distinction between it and the medulla oblongata—takes place on a level with the first cervical pair of nerves, consisting chiefly in the decussation of the pyramids.

Large bundles of fibrils collect from the posterior division of the lateral columns, and pass obliquely forward and upward, and simultaneously forward and inward towards the anterior longitudinal fissure. They appear here more or less as continuous bands, though frequently interrupted by vertical fibrils. They cross the fissure, and then run up the other side in the place of the anterior columns, which they have now displaced. There is likewise a considerably enlarged anterior commissure, here called the decussation. The bundles of the pyramids too divide the anterior gray columns from the central gray substance, and displace them outward (Fig. 22).

These decussating fasciculi are to be regarded as chiefly motor. They form the coarse-bundled decussation which we see in the anterior fissure, and are called *the inferior or motor decussation of the pyramids.*

Higher up too we may perceive bundles of quite considerable dimensions, arising from the posterior columns, bending forward, passing the central canal, crossing the middle line, and finally running up in the pyramid of the opposite side, where they generally occupy the exterior portion. This is the *anterior* or *sensory decussation*. Huguenin describes a bundle from the posterior cornua as taking part in this, but Flechsig's latest investigations have, to say the least, thrown great doubt upon the significance of these fibres and the exactitude of their course. Flechsig positively denies any connection whatsoever between this anterior decussation and the pyramids proper. Still, these fibrils most undoubtedly alter the configuration of the gray substance in the spinal cord, dividing the head and neck of the posterior gray cornua from the central gray substance (Fig. 22). At the same time, these parts are displaced somewhat outwardly towards the periphery, since the lateral columns diminish in size. The increase of volume in the posterior gray columns enables us to follow them on transverse sections through the greater part of the medulla and in their gradual outward course (cf. Fig. 22, *b*, Figs. 24–26, *c*). In the peripheral portion of these sections we observe large bundles of fibrils ascending vertically; these are the large *ascending roots of the trigeminus* (Fig. 22, *b*), which may be followed down to the second and third cervical nerves and upward as far as the exit of the sensory trigeminal roots.

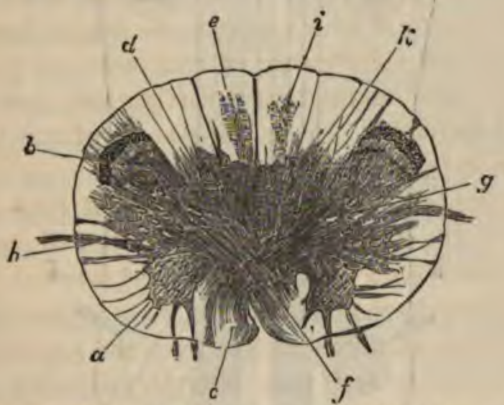

FIG. 22.—Transverse section through the inferior portion of the medulla oblon. at the level of the decussation of the pyramids. *a*, Anterior gray columns; *b*, head of the posterior column and ascending root of the trigeminus; *c*, anterior columns (pyramids); funiculi, *d*, cuneati, et *e*, graciles; *f*, decussation of the pyramids; *g*, formatio reticularis; *h*, roots of the accessorius; *i*, nucleus of the gracilis; *k*, nucleus of the cuneiform.

The *motor decussation* has lately been submitted to thorough investigations, and appears to be derived mainly from the postero-lateral columns. The diagram (Fig. 23) enables us to see this, and observe how the fibrils, before crossing the middle line, bend gradually forward, and how they displace the anterior columns after their passage across. The figure demonstrates too the bundles from the anterior columns which join the pyramids on either side and pass up along with these *undecussated* (Flechsig). Thus we see that the pyramids contain not solely the decussated fibrils of the postero-lateral columns, but also some additional undecussated elements, varying in number, from the anterior columns. Flechsig positively denies the association with the pyramids, at an anterior point, of bundles from the posterior columns and cornua, though it has been asserted that this takes place, and that these run with the pyramids into the pons.

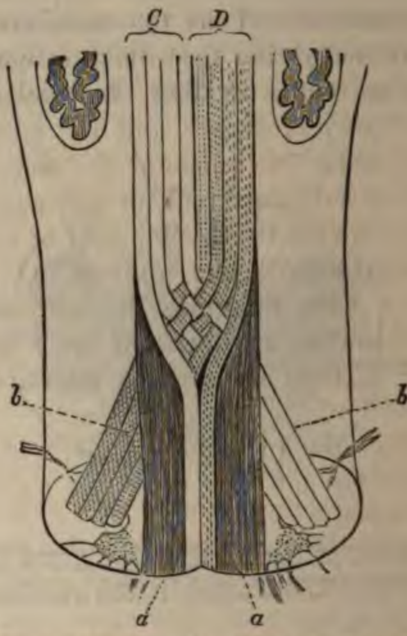

FIG. 23.—Diagram of the motor decussation. Transparent longitudinal view of the medulla, with only the anterior and the decussation of the postero-lateral columns left. *a*, *a*, Anterior columns; *b*, *b*, pyramido-postero-lateral columns; the pyramidal columns of the right side (*c*), arising from the left lateral column and reinforced by a slender bundle from the right anterior column, are left unshaded, those of the left side are dotted. The part of the anterior columns which disappears behind the pyramids is shaded with vertical lines.

The deflection of so many bundles from the lateral and posterior columns produces a number of fibrils, which lie horizontally on a transverse section, completely interwoven, and embracing in their meshes the vertical fibrils, after these latter have split up in every direction. Thus from the reticular processes of the spinal cord we get the *formatio reticularis* (Fig. 22, *g*). This latter structure occupies more and more space in the transverse section as we follow it upwards, and connects all the fibrils and structures of the medulla. But its complicated mesh-work is scarcely to be unravelled. Thus the transverse section even of the inferior portions of the medulla assumes the altered appearance we see in Figure 22. The four cornua are separated from the central gray substance, and the posterior are further very much dislocated. The centre of the section is occupied by the remaining gray substance and an enormous formatio reticularis. In addition, we see the development of new gray substance around the different bundles and columns. On particularly good sections one may often recognize the horizontal fibrils of the roots of the accessorius, arising from the neighborhood of the lateral cornua, *i. e.*, from the tractus intermedio-lateralis (Fig. 22, *h*).

Higher up, while the formatio reticularis is visibly increasing in size, we are met by new structures, the significance of which is only in part known.

Of these structures the most striking are the *olivary bodies* (Figs. 24, 25, 26, *b*). They arise in the territory of the lateral columns, close upon the pyramids, and attain the length of 14 mm. They resemble an extremely irregular disc, and contain a number of small ganglion-cells. On the whole, they look like a bean or an almond, with the hilus directed inwards. A number of nerve-bundles, coming from all points, enter at this hilus, some terminating in the gelatinous substance of the olivary bodies, while others pass through in the form of fine bundles. This appears to bring about an extremely wide connection with all the different parts of the medulla oblongata.

Further on we perceive here a number of gray nuclei, most of which are connected with some cerebral nerve, and must be regarded as medullary roots. The first of these is the *hypoglos-*

sal nucleus (Figs. 24, 25, 26, *g*), situated beside the middle line, and falling back with the central canal. At the entrance of this canal, just below the floor of the fourth ventricle, we find it lying on the edge of the median fissure.

This nucleus attains the length of about 18 mm.; it is cylindrical, attenuated, and contains a number of large ganglia, resembling in every respect the multipolar cells of the anterior cornua. It corresponds exactly to the anterior cornu, though it retreats along with the central canal, which opens posteriorly, while an increasing number of fibrils pass over into the anterior portion of the medulla. Thus the anterior end of the hypoglossal nucleus comes to be situated on the floor of the fourth ventricle. The roots of the hypoglossus (Figs. 24 and 25, *XII*) run horizontally between the pyramids and the olivary bodies to join their nucleus, and their whole course is easily followed.

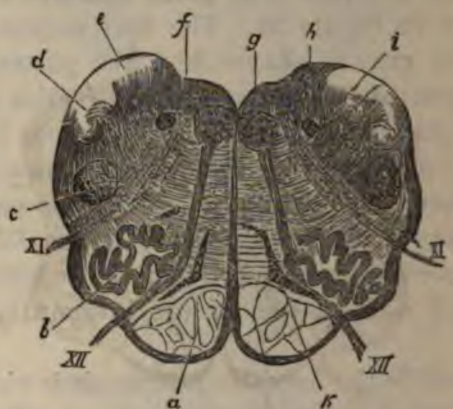

FIG. 24.—Transverse section of the medulla through the inferior portion of the olivary body and the superior termination of the nucleus of the accessorius. *a*, Pyramids; *b*, olivary body; *c*, gelatinous substance, and ascending root of the trigeminus; *d*, corpus restiforme; *e*, funiculi cuneati, f. graciles; *f*, nucleus accessorii; *g*, nucleus hypoglossi; fasciculus rotundus (respiratory fascicle of Krause); *i*, nucleus funiculorum gracilium et cuneatorum; *XI* and *XII*, radices accessorii et hypoglossi; internal parolivary bodies (nucleus of the pyramids).

Somewhat posterior to the nucleus hypoglossi, and in the inferior portion of the medulla, we find the *nucleus of the accessorius* (Fig. 24, *f*). It, too, is represented by a columnar, attenuated, gray structure, which extends to the tractus intermedio-lateralis of the spinal cord (cf. Part I., p. 16), and with this it gradually alters its position in relation to the other parts of the section. From it the superior roots of the accessorius run laterally and somewhat posteriorly.

The nucleus accessorii is immediately bounded by a gray mass, which is situated in this region, beside the nucleus hypoglossi. This is the nucleus of the *vagus*. It contains a number of small and middle-sized ganglia, and lies in the fourth ventricle, along with the hypoglossal nucleus, in the so-called ala cinerea, immediately beneath the surface.

Immediately adjacent to the internal portion of this nucleus comes a clearly defined, round fascicle of coarse fibrils (Figs. 24 and 25, *h*). We can trace this along the whole course of the nucleus vagi, and find it composed of fascicles from the vagus, glossopharyngeus, and accessorius. It runs down into the cervical portion of the cord, and is said to come into connection with the phrenic (Krause's respiratory fascicle). According to Stieda, the roots of the three above-mentioned nerves are derived from this lateral ascending longitudinal fascicle, and *not* from the gray structures, which are generally designated as their nuclei.

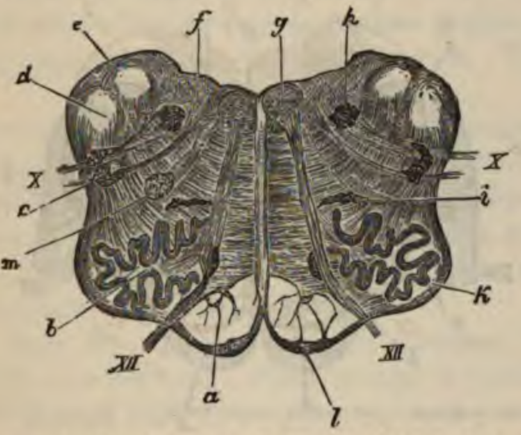

FIG. 25.—Transverse section through the centre of the olivary body, and of the vagus-root. *a*, Pyramidal fasciculi; *b*, olivary body; *c*, gelatinous substance and trigeminus-root; *d*, corpus restiforme; *e*, funiculi graciles et cuneati; *f*, nucleus vagi; *g*, nucleus hypoglossi; *h*, fasciculus rotundus (respiratory fascicle); *i*, parolivary body (external); *k*, fibræ arciformes; *l*, nucleus of the same; *m*, motor nucleus of the vagus (Meynert); *X*, *XII*, fasciculi of the vagus and hypoglossal roots.

The nucleus of the vagus is bounded, too, by another gray structure, which comes into immediate contact with it, and is not separated by any distinct boundary ; it lies, however, somewhat less superficially, and is designated as the *nucleus of the glossopharyngeus* (Fig. 26, *h*). The roots of the glossopharyngeus run almost horizontally from it, outwards and forwards, making their exit from the medulla above the roots of the vagus.

Meynert and Huguenin describe further a motor nucleus of the vagus and glossopharyngeus, which is situated more in the interior, between the olivary body and ascending root of the trigeminus. We have indicated its position in Fig. 25 by *m*. Stieda looks upon it as the nucleus proper of the vagus.

The *posterior median nucleus of the acusticus* (Fig. 26) (nucleus acust. sup. of Henle; median nucleus of the posterior root of the acusticus of Krause) comes likewise into contact with the vagus; but it is more superficially situated, and somewhat to the outer side of the glossopharyngeal. It occupies the whole space included between the ala cinerea and pedunculus cerebelli, up to the anterior border of the striæ medullares. The posterior root of the acoustic takes its chief origin from this nucleus, and passes out partly in superficial fasciculi (striæ acusticæ), and partly through the body of the medulla. Between these two fasciculi we find the *posterior lateral acoustic nucleus* (Fig. 26, *i*) (nucleus acust. inferior of Henle) lying beside the peduncle, in the form of a small gray nodule. Besides these, the acoustic possesses two other nuclei, belonging to its anterior roots, and

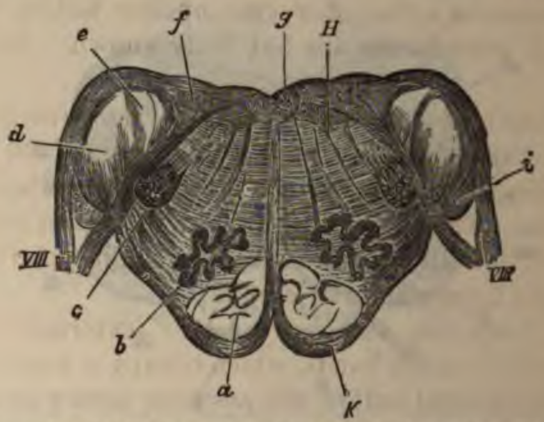

FIG. 26.—Transverse section of the medulla through the superior end of the olivary body, where the posterior median acoustic nucleus occurs. *a*, Pyramids; *b*, olivary body; *c*, gelatinous substance and ascending trigeminus-root; *d*, corpus restiforme; *e*, funiculi cuneati et graciles; *f*, posterior median acoustic root; *g*, anterior termination of the hypoglossus nucleus; *h*, glossopharyngeus nucleus; *i*, posterior lateral acoustic nucleus; *k*, fibræ arciformes; *VIII*, posterior acoustic roots.

situated in that portion of the medulla which lies anterior to the striæ. The first is the *anterior median acoustic nucleus* (Fig. 29, *VIII*) (median nucleus of the anterior acoustic roots of Krause), and occupies the external angle of the fourth ventricle, about the middle of the pedunculus cerebelli. The second appears like a prolongation of the postero-lateral acoustic nucleus beside the peduncle, and is wedged in between the peduncle, the crus cerebelli ad pont. and the flocculus. It receives the name of *anterior lateral acoustic nucleus* (nucl. acust. lateralis of Henle), and gives origin to the portio intermedia Wrisbergii.

We have now described in the medulla oblongata, below the level of the pons, all the structures that are at all well known. There are, however, a few other gray nuclear bodies, whose signification and connections are but little known. These are the following:

The *nucleus of the pyramids* (*internal parolivary body*, Fig. 24, *k*), a structure which is bent at a sharp angle, and runs vertically, consisting of gray substance of the same structure as the olivary bodies. They lie opposite the pyramids in front, and to the inside of the olivary bodies, corresponding nearly with the position of the former anterior cornua.

Then we have the *parolivary bodies* (external, Fig. 25, *i*), similar, slightly concave bands, which occupy a position exactly bounding the internal half of the posterior border of the olivary bodies.

Further, we find the *nucleus of the cuneiform column* arising from what was the anterior cornu, a longish, gray body on the inner border of the cuneiform column, and enlarging as it ascends (Fig. 22, *k*, Fig. 24, *i*).

Fourthly comes the *nucleus of the funiculus gracilis*, in the shape of a longitudinal pillar of gray substance. It arises from the centre of the funiculus gracilis, and produces the enlargement in this, known as the clava (Fig. 22, *i*, Fig. 24, *i*). It ascends with the nucleus of the cuneiform column, and both extend to the posterior end of the postero-lateral acoustic nucleus.

The last on this list is the *nucleus of the lateral column*, a small, cylindrical, attenuated column of cells in the posterior portion of the lateral column between the olivary body and the caput cornu posterioris.

The remainder of the transverse section is occupied by the *formatio reticularis*, which attains its maximum dimensions and symmetry parallel with the olivary bodies. This structure is constituted of a number of fascicles, which run horizontally in an arched course, though sometimes radially, and embrace in their meshes a number of vertical bundles. All through its substance we see numerous ganglia, so that we find the most intimate intermixture and intertwining of white and gray substance. The place of the anterior commissure below the decussation of the pyramids is filled above this point by the *raphe*. This structure enlarges in its antero-posterior axis as it ascends, and consists both of longitudinal and transverse fibres. We have further to mention the *fibræ arciformes*, which surround almost the whole outer surface of the medulla in the form of a layer (varying in dimensions) of fibres that take a horizontal or an oblique course. They increase in size as they ascend, and generally run into the raphe (Figs. 25 and 26, *k*).

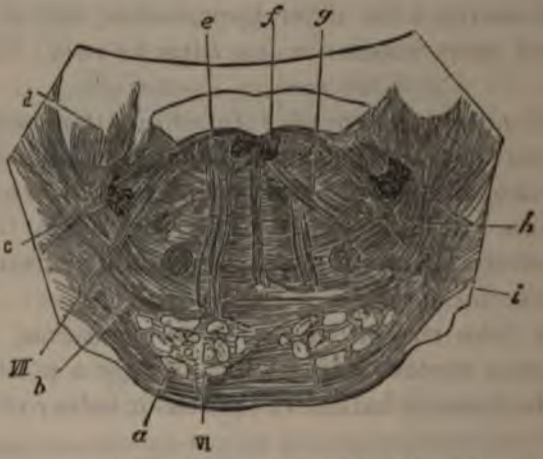

FIG. 27.—Transverse section of the medulla through the inferior border of the pons, on a level with the facial and abducens roots. Somewhat diagrammatic. The right half represents a section made a little below the left section. *a*, pyramids; *b*, transverse fasciculi of the pons; *c*, gelatinous substance and ascending trigeminus root; *d*, pedunculus cerebelli; *e*, nucleus of the abducens (nucleus common to the facialis and abducens); *f*, genu nervi facialis; *g*, descending limb of the facialis root; *h* nucleus of facialis; *i*, anterior olivary body; *VI*, root-fascicle of abducens; *VII*, do. of facialis.

Almost parallel with the most anterior striæ we are met on the anterior surface of the medulla by an enormous body of transverse fasciculi, *i. e.*, the *pons Varolii* (Fig. 27, *b*, Fig. 28). It is this structure which produces the characteristic appearance of the anterior portion of the medulla. The pons consists of numerous and well-developed transverse bundles, between which we can trace scarcely anything but the main longitudinal fibres, namely, the pyramids.

The most important and best-studied parts of this anterior portion of the medulla are the nuclei of the fifth, sixth, and seventh cerebral nerves.

The best and longest known of these is the *nucleus of the abducens* (mixed nucleus of the facialis and abducens, Fig. 27, *e*). It lies in the continuation of the hypoglossus, only divided from this by a very short interval, and occupies a position just above the striæ on the side of the median fissure, which extends about six or seven millimetres in the form of an attenuated gray column. The roots of the abducens run almost horizontally from the pyramidal region towards the pons, at the inferior border of which they diverge slightly.

This nucleus appears, too, to be connected with the facialis, for the facialis root takes its course from the lateral region of the pons towards the median fissure of the rhomboid sinus, close to the abducens-nucleus. From the descriptions of Henle and Huguenin, there can be no doubt that the facialis root gives off a number of fibres which pass into this abducens-nucleus; hence it has been designated as "posterior facial nucleus." After this, the facial root takes a sudden backward turn, and runs for a short distance parallel to the median fissure. Turning again sharply (genu nervi facialis, Fig. 27, *f*) it presses forward and into the interior of the medulla, where it meets a longish fascicle of gray substance, the *nucleus proper of the facialis* (Fig. 27, *h*). Thus we see the root of the facialis only passes round the abducens nucleus in a horseshoe course, and is certainly in but very small part derived from this nucleus. The latest observers return to the old view, and deny even the remotest connection between the facialis and the nucleus of the abducens.

The question about the facial nucleus does not appear to have received its final answer. Amongst others, Lockhart Clarke describes a so-called "inferior" facial nucleus in the inferior portion of the medulla on a level with the hypoglossal and accessorial nuclei, and indeed pathological facts (bulbar paralysis) seem to confirm its existence.

In connection with the trigeminus, the complication is still greater. We have already described one of its roots, the *ascending*, which belongs to the caput cornu posterioris, and may be traced down through the whole length of the medulla into the cervical region of the spinal cord. So we are justified in regarding the caput cornu post. as *inferior sensory trigeminal nucleus* (Fig. 23, *b*, Figs. 24–28, *c*). The *motor trigeminal nucleus* is situated on the floor of the fourth ventricle, close to the upper border. Its anterior termination coincides with the entrance of the Sylvian aqueduct, and from this it extends in form of a fine, gray column to the border of the facialis roots. This nucleus gives origin to the portio minor of the trigeminus. At the point where the roots of the portio trigemini major separate, and run some forward, and the rest backward, we find a small gray mass lodged between the two, representing the *middle sensory nucleus of the trigeminus* (Fig. 28, *f*). The smaller portion of root-fascicles (Fig. 28, *b*), which takes an anterior course, passes to the far side of the corpora quadri., and terminates there in the

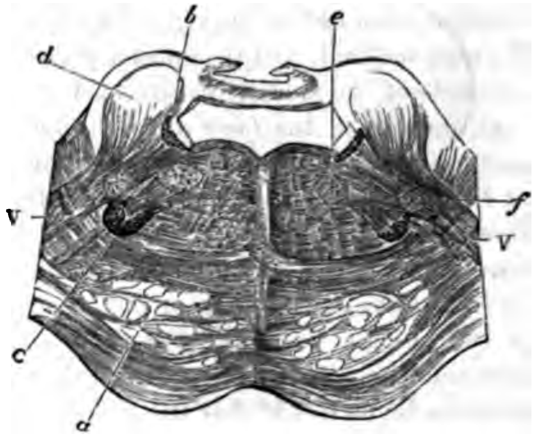

FIG. 28.—Transverse section of the medulla through the anterior portion of the fourth ventricle, coinciding with the exit of trigeminus. Semi diagrammatic. *a*, pyramids; *c*, gelatinous substance and ascending root of trigeminus; *b*, descending trigeminal root, of cerebral origin; *d*, crus cerebelli ad corp. quadr.; *e*, motor nucleus and root of trigeminus; *f*, middle sensory trigeminal nucleus; *V*, root-fascicle of trigeminus.

superior sensory trigeminal nucleus. Thus we can count up four nuclei for the trigeminus, as we did for the acoustic.

In addition to these gray nuclei we meet in the superior portion of the medulla: 1, the *superior olivary body* (27, *i*), a longish, gray column, which is situated in the pons just in front of the facial nucleus, and 2, the *pons nuclei*, consisting of patches of gray substance scattered through the whole pons.

The remainder of the transverse section through the anterior part of the medulla is taken up by the transverse fibres of the pons, and the formatio reticularis.

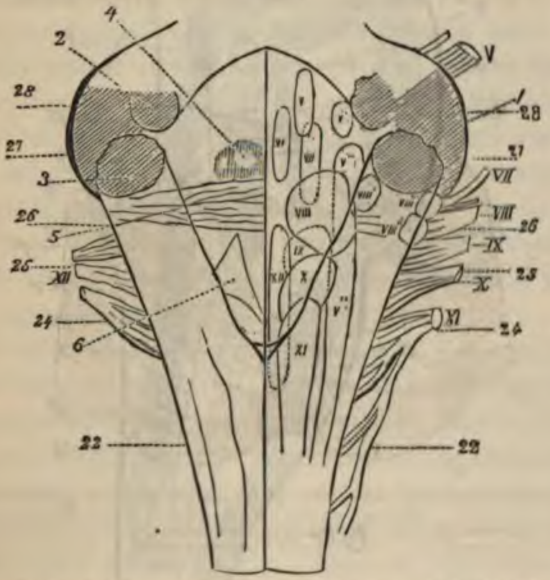

FIG. 29.—Transparent view of the medulla from behind. On the right hand the nerve-nuclei are diagrammatically represented, and lettered with Roman numbers. *V*, motor trigeminal nucleus; *V'*, median, and *V''*, inferior sensory trigeminal nuclei; *VI*, nucleus of abducens; *VII*, facial nucleus; *VIII*, posterior median acoustic nucleus; *VIII'*, anterior median, *VIII''* posterior, and *VIII'''*, anterior lateral acoustic nuclei; *IX*, glossopharyngeal nucleus; nuclei of *X*, vagus, *XI*, accessorius, and *XII*, hypoglossus. 1, crus cerebelli ad pontem; 2, crus cerebelli ad corp. quadr.; 3, crus cerebelli ad medullam; 4, eminentia teres; 5, striæ acusticæ; 6, ala cinerea. The Roman numerals (V.—XII.) beside the figure represent the corresponding nerve-roots. The Arabic numerals (22, 24–28) are attached to the transverse section of roots, which bore the same numbers in foregoing figures.

It is evident from the complicated relations between all the structures we have been describing that it is no easy matter to get a correct idea of the topographical anatomy of the fresh unhardened organ, one that will take in every particular, and enable us at a post-mortem examination to say without hesitation what nuclei or nerve-fascicles, etc., coincide with some small patch of softening, some diminutive apoplexy, or with some defined sclerotic centre. In order to lighten the practitioner's task in this direction, we insert two diagrammatic sketches, which give lateral and front views of the approximate situation of the most important gray centra. We here follow the example of others, such as *Stilling*, *Krause*, et al. These diagrams do not of course make the very slightest pretensions to extreme accuracy, but still we hope they will give an easily comprehensible picture of the topography, and one that will act as a key to this anatomical labyrinth.

Fig. 29 represents a transparent view of the medulla oblongata from behind, on the right side of which all the above-mentioned nuclei are represented and their names attached, while on the left side, to aid comparison, nothing is given but the superficial structures, such as the ala cinerea, striæ medullares, etc. By this means one sees plainly the nuclei of the sixth and seventh, of the fifth and eighth, the hypoglossal nucleus, and the inter-connected nuclei of the ninth, tenth, and eleventh pairs. Of course, neither the form nor size of the diagrammatic nuclear structures is exactly true to nature, the only thing that is aimed at being a faithful representation of their situation as relates to one another and the surface. Further, we have sacrificed a number of details to the clearness of the sketch.

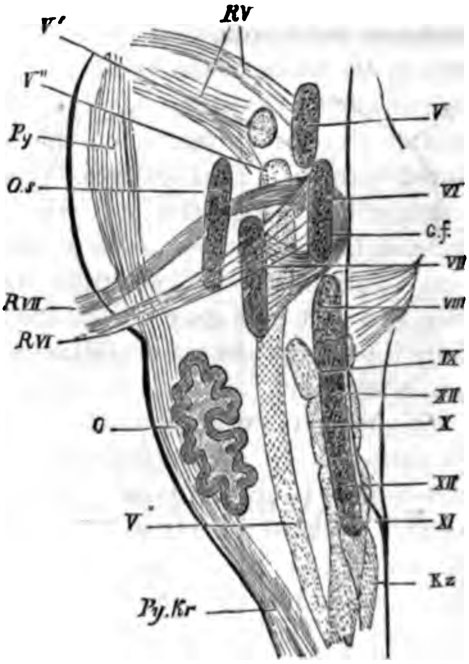

FIG. 30.—Transparent lateral view of the medulla, showing the relative positions of the most important nuclei; right half of the medulla, seen from the surface of section; the parts that lie closer to this surface are deeper shaded. Diagrammatic. *Py*, pyramidal tract; *Py Kr*, decussation of pyramids; *O*, olivary body; *Os*, superior olivary body; *V*, motor, *V'*, middle sensory, *V''*, inferior sensory nucleus of trigeminus; *VI*, nucleus of abducens; *Gf*, genu facialis; *VII*, nucleus facialis; *VIII*, posterior median acoustic nucleus; *IX*, glossopharyngeal nucleus; *X*, nucleus of vagus; *XI*, accessorius nucleus; *XII*, hypoglossal nucleus; *Kz*, nucleus of the funiculus gracilis; *R V*, trigeminus root; those of the *R VI*, abducens and *R VII*, facialis.

Fig. 30 gives a transparent view of the right half of the medulla, seen from the surface of section (*i. e.*, antero-posterior, through the middle line, and vertical), so that what is nearest to the observer in the diagram is nearest to the middle line. In this figure, too, only some of the gray nuclei are represented, in order clearly to display their relation to the exterior and their internal relations of space. The nuclei nearest the observer (*i. e.*, nearest the middle line) are deeper shaded, while those that are further from him, and consequently nearer the surface of the medulla, have only a light shading. Amongst the nerve-tracts which communicate with the nuclei we have given only semi-diagrammatically those of the fifth, sixth, and seventh nerves, especially the latter, in order to render the genu nervi facialis more distinct. Only the three nuclei of the trigeminus, which lie within the medulla, are sketched, and only the principal nucleus of the acoustic.

Connection between the histological elements, and the course of the nerve-fibres in the medulla oblongata.

Notwithstanding all the investigations that have been made and the incalculable trouble expended on this subject, we are scarcely acquainted with the barest outlines. We know just as little about the fate of most of the fasciculi, which enter the medulla from below and from above, as we do of the significance of numerous striking structures in the medulla. Their connection with one another and with the gray nuclei, the mode of interruption and where it occurs, their transposition and their termination, all remain a mystery.

Still we know somewhat more about the *course of the root-fibres* from their entrance into the medulla till they reach their proper nuclei, where they temporarily end. The root-fascicles of the hypoglossus, and perhaps, too, those of the abducens, are the only ones which run directly and almost horizontally from their point of entrance, and can be followed on a transverse section along nearly their whole course. Most of the other root-fibres take a more or less oblique course and are often tortuous. Partly, indeed, their course is comparatively simple; but sometimes they break up into separate fascicles, and sometimes press inwards towards the nuclei in more compact masses; such are the root-fascicles of the accessorius, vagus, and glossopharyngeus. But others are much more tortuous, and split up in all directions to join nuclei that lie far apart; to this category belong the roots of the trigeminus and acoustic. The roots of the facial, finally, as we described them above, form a large arch with its crown (genu) touching the posterior surface of the medulla, and its pillars in the facial nuclei.

There have been many investigations made, and much discussion, on the possibility of a *decussation* of the root-fibres in the medulla. It seems, however, to be settled beyond doubt, that *by far the greater part* of the fibres of every nerve that enters the medulla remains on the same side, and ends in the gray nuclei of the side. The decussation of some few fibres has been asserted, but by no means proven. Krause's words on this subject represent truly the present extent of our knowledge; he says: "The roots of the abducens, of the portio trigemini minor, the anterior acoustic and the inferior accessorial roots most certainly do not decussate; a partial decussation (of some fibres) of the portio trigemini major, of the posterior acoustic roots, of the glossopharyngeus and vagus may take place; *possibly*, too, of the facial, the superior accessorial, and the hypoglossal, but the assumption of the partial decussation of these latter may be due to misconceptions." In any case the decussation of all these tracts (and we are compelled by a number of clinical facts to assume their decussation) takes place after they have passed their nuclei, further up in the medulla or in the pons. But the exact place where it occurs is totally unknown, since even the central continuation of all these nerves after they pass into their nuclei is almost completely a blank to us. We can only conjecture that these tracts are to be looked for in the formatio reticularis, and that they then successively cross in the raphe, and afterwards join other tracts (*e. g.*, the pyramidal) at different points as these come up from the spinal cord to the brain.

Our knowledge, too, is at the same low point as regards the course of the different tracts into which we can dissect the spinal cord, and as regards the ultimate course of the spinal columns in the medulla oblongata.

The course of the pyramidal tracts has, however, been settled beyond all doubt by the thorough investigations of Flechsig. They are collected from the posterior portions of the lateral and the internal fasciculi of the anterior columns, and after the decussation of the parts alone derived from the lateral columns, they run directly and continuously through the medulla upwards; they then enter the pons, and separate gradually into a number of fibrils, but without any transposition or discontinuity, till they arrive at the pedunculus cerebri, where they pass through the inner capsule and further on into the brain. In the pons the pyramids appear to be reinforced by fibres from the pons and fibræ arcuatæ, which probably come from, or are going to, the cerebellum. Flechsig positively denies any addition to the pyramids from the so-called sensory decussation of the posterior columns and cornua.

The *direct tractus ab cerebello ad columnas laterales*, which Flechsig, too, has studied so accurately, runs completely into the restiform body, though it is only one factor in this structure. The two go together into the cerebellum, where their termination has not yet been discovered. The real significance of this tract has not been determined, but it may have some connection with Clarke's columns.

It is a more difficult matter when we come to the *posterior columns*. The so-called *funiculi graciles* (Goll) probably end in great part in the "nuclei of the columnæ graciles" at the level of the clavæ. In the same way the greater part of the funiculi cuneati terminates in the nucleus of this column; *possibly*, however, some of its fibres go directly as fibræ arcuatæ into the formatio reticularis, olivary body, etc. But the further course of the posterior columns is totally unknown. The old view (which rested on external appearances) that the funiculi graciles et cuneati continued straight into the pedunculi cerebelli, and ran with these into the cerebellum, while the external portion of the pedunculi (the corpus restiforme proper) was derived through the fibræ arcuatæ ext. et int. from the antero-lateral column—this view was most decidedly a false one. But the modern view, which has been worked out chiefly by Meynert, has not passed without opposition. Meynert considers that the greater part of the posterior columns (or their continuation) runs into the olivary and parolivary bodies of the same side, and that then their continuations pass across the raphe, finally forming the restiform body of the opposite side. Accordingly we should have the virtual continuation of the spinal posterior columns in the restiform bodies of the opposite side, and the olivary and parolivary bodies thrown into their course. What are defined as funiculi graciles et cuneati in the pedunculi cerebelli, and which lie in the middle line, these Meynert describes as structures of new origin and springing from the formatio reticularis, which higher up join the spinal columns of the same name, and take their place, without being continuations of these, in the same manner as the pyramids take the place of the anterior columns.

Space does not allow us to give in detail Flechsig's objections to this view of Meynert's. We shall only mention that Flechsig describes a great number of fibres which come from the nuclei of the funiculi graciles (anterior decussation) and accompany the pyramids for some distance. They soon, however, leave the pyramids, and rejoin the formatio reticularis in the neighborhood of the olivary and internal parolivary bodies. Flechsig, too, asserts the existence of an extensive connection between the olivary body and the cuneiform column of the same side. According to the same author at least a portion, the outer portion, of the restiform body is composed of fibres, which spring from the formatio reticularis and the region of the olivary decussation.

The *remainder of the anterior*, after deduction of the pyramido-anterior columns (Flechsig's fundamental anterior column) runs directly upwards into the so-called posterior longitudinal fascicle of the medulla, which is situated posteriorly between the roots of the hypoglossus and the raphe. This fascicle is then continued through the posterior portion of the pons into the cerebrum.

What remains of the lateral columns, when we put aside the pyramido-lateral tract, is lost in the formatio reticularis, and is here possibly in direct connection with the corpora quadrigemina and the thalamus opticus. From the latter two structures, as we must not forget, a very considerable number of fibres descend to the medulla, and take part in the construction of the formatio reticularis. In that portion of the formatio reticularis which lies between the olivary bodies, Flechsig describes the fibres that are connected with the corpora quadrigemina, and in a region of the format. retic. posterior to this he places the fibres which come from the optic thalamus.

After we have exhausted all that we know perfectly and in part in the medulla, there remains a vast field of which we know nothing whatsoever. The exact connections and significance of the olivary and parolivary bodies, the pyramidal nuclei, etc., are all totally unknown; further, we know nothing of whence the innumerable fibres of the substantia reticularis come, and whither they go. We are equally ignorant of the central tracts of all the different bulbar nerves, and of the ending and significance of the fibres of the pons. Nor are we acquainted with the origin and function of the majority of the peduncular tracts. Further, none of the centra, the existence of which has been assumed to explain physiological facts, the centra for the respiratory and cardiac movements, and for deglutition, the vaso-motor centra and the centrum for convulsions—in fact, all the great reflex tracts of the medulla—none of these has been recognized by the anatomists. Doubtless the formatio reticularis does display the most suitable anatomical structure for the accomplishment of every possible reflex action, and so it is perhaps allowable to *conjecture* that this formation chiefly serves for reflex processes; but there is an extremely wide gulf between a conjecture like this and a certainty which clears up every detail.

We consider it advantageous to give a diagram of the course of the fibres (after Flechsig, Arch. der Heilk. XVIII. Taf. VI.) to enable the reader to get a general conception of the subject, and at the same time show him how complicated the study is. Figure 31 represents the medulla, pons and the neighboring structures, leaving out all the gray nuclei except the olivary bodies, and only giving the formatio reticularis as a shaded space. The sketch gives a general view of the most important and best-known tracts, which ascend from the anterior, lateral, and posterior columns of the spinal cord (*V*, *S*, *H*). We can see plainly how the pyramidal tracts (*Py*) are formed from the pyramido-anterior (*a*) and pyramido-lateral (*d*) tracts. And then we can follow the course of the cerebello-lateral columns (*e–e*) which ascend to the pedunculi cerebelli. Then we observe how the internal portions of these pedunculi (*P.c.i*) are lost in the formatio reticularis, whilst the external (*P.c.e*) run into the olivary bodies. The remainder of the anterior (*b*) and lateral (*c*) columns end in the formatio reticularis. The fibres of the funiculi graciles (*f*), after piercing the nucleus of the funiculus (*f'*), form the anterior decussation of the pyramids, join the pyramidal tracts, and then immediately fall back into the olivary bodies and the formatio reticularis. The tracts of the cuneiform column (*g*) after they reach their nuclei, spread out into the olivary bodies, and partly into the formatio reticularis. This formation further receives fibres from the thalamus opticus (*Thal.*) and corpora quadr. (*C.q.F*). From the latter structures, too, fibres pass into the olivary bodies (*C.q.O*).

Thus we see that the formatio reticularis receives fibres from every quarter, and that these fibres are here woven into a complete chaos of threads. If we only imagine the addition of a number of other gray nucleolar structures (to which we have here made allusion), and all their afferent and efferent fibres, the root-fibres, the fibræ arcuatæ, the fibres of the pons, etc., we shall then have a painfully acute idea of the structure with which we have to deal, and the hopelessness of ever unravelling it; we shall then, too, comprehend how our knowledge of the finer structure of the medulla has remained so extremely incomplete, in despite of all the work and trouble bestowed upon it.

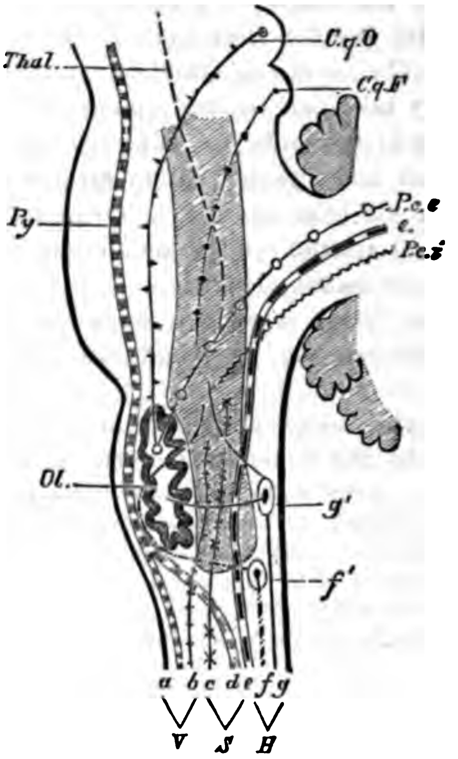

FIG. 81.—Diagram of the chief tracts in the medulla. The formatio reticularis is represented by shading. *Ol*, olivary body; *V*, anterior, *S*, lateral, and *H*, posterior spinal funiculi: *a*, pyramido-anterior tract: *d*, pyramido-lateral tract; *Py*, pyramidal tract; *b*, remainder of anterior column; *c*, remainder of the lateral column; *e–e*, cerebello-lateral tract; *f*, funiculus gracilis, and *f'*, nucleus of the same; *g*, funiculus cuneatus, and *g'*, nucleus of the same; *P.c.i*, internal fasciculus of the peduno. cerebelli: *P.c.e*, external fasciculus of the same; *C.q.F*, tract from corp. quadr. to format. retic.; *C.q.O*, the same to the olivary body; *Thal.*, tract from the thalamus opticus.

Vessels of the medulla oblongata.—Duret's exhaustive investigations have proved that the medulla gets nearly its whole supply of blood from the *vertebral* and *basilar* arteries, the latter supplying the pons portion. Here we have chiefly to mention small, *median* branchlets which pierce into the raphe between the pyramids, and are given off by the art. spin. ant. for the bulbus and by the basilaris for the pons. They run up to the floor of the fourth ventricle, and ramify here, forming a large, narrow-meshed capillary network, especially round the gray nuclei. Thus we see that they originate chiefly from the left vertebral, for this artery generally gives off the art. spinal. ant. when it is single, and when there are two anterior spinal arteries the median bulbar branchlets are generally derived from the ant. spin. sinistra. But besides these, *small vessels* pierce the medulla *along with the nerve-roots*, and run to the corresponding nuclei, where they take part in the formation of capillaries. The olivary bodies and pyramids are supplied by small branches from the vertebral and anterior spinal arteries. The restiform bodies receive vessels from the art. cerebelli infer. post., whilst the posterior spinal arteries send branchlets to the pedunc. cerebelli and floor of the fourth ventricle.

Most of these little vessels are so-called terminal arteries (Cohnheim's "*Endarterien*"). Their ramification produces a comparatively open-meshed network in the white substance, while in the gray the meshes are very numerous and narrow. It need hardly be said that numerous varieties and abnormalities of origin and course occur amongst these little vessels, and these are of some importance in the explanation of certain clinical occurrences.

There is nothing special to be said of the *veins*, and the *lymph-vessels* in the medulla are quite analogous to those of the spinal cord.

II. Physiological Introduction.

Amongst others we would direct attention to: *Longet*, l'Anatomie et physiologie du système nerveux. T. I.—*Schiff*, Lehrbuch der Physiologie des Menschen. I. 1858-59.—*Schroeder v. d. Kolk*, Bau u. Function der Medulla spin. und oblong. 1859.—*Wundt*, Physiol. Psychologie. Leipzig. 1874.—*Laborde*, Dictionn. encycl. des sc. méd. 2. sér. T. VIII. p. 604. 1874.—*Poincaré*, Le système nerveux central, etc. 2me édit. T. I. 1877.

Besides these, the text-books of *L. Hermann*, *Wundt*, *Funke*, *Vierordt*, etc., may be consulted, and innumerable monographs in all possible physiological periodicals.

The physiology of the medulla oblongata is just as imperfect and obscure as its anatomy, so that we have not become possessed of any undisputed information on the most elementary and important facts. For instance, we know nothing of the conduction of sensory impressions, or of the exact position of the chief centres.

We are, indeed, acquainted with a number of facts, which teach us to recognize the medulla as one of the most important divisions of the central nervous system, and which give it a prominent part in several all-important physiological processes, such as respiration, the cardiac movements, vascular innervation, speech, and others. But we have got little beyond the confirmation of these facts, and we are in especial darkness as to the anatomical distribution of these functions and their components.

There are very good reasons for all this, and the chief of them is the difficulty of experimentation. For the parts lie so deep and are so well protected, the slightest injury to them is so dangerous, the structures themselves are so extremely small, they are so wonderfully connected in a complicated manner with all possible adjacent structures, and the white and gray substances are so intimately mixed—all these circumstances are so many insurmountable difficulties in the way of physiological investigation.

Consequently we must not claim too much from physiology. We should undoubtedly look for most important explanations from careful clinical observation, combined with accurate post-mortem examination, though this has yet been scarcely attempted.

In this state of things we can and must give the practitioner only a very brief account of the physiology of the medulla oblongata.

We shall first study the *medulla as an organ of conduction.* Nobody can entertain the slightest doubt that the motor impulses sent from the cerebrum to the spinal cord, and the sensory irritations on their way from the periphery to the brain, must pass through the medulla oblongata. Further, we are justified in assuming that the parts of conduction for several other irritations, which pass between the spinal cord, the cerebrum and cerebellum, must likewise be situated in the medulla. But the position of these has not yet been properly demonstrated.

Motor Conduction.

The investigations in this direction have gone further than any, and, indeed, have reached a certain degree of completeness. It is almost beyond doubt that *the chief paths of conduction for voluntary motor impulses are to be sought in the pyramidal tracts* (Flechsig). We should thus have the chief conductors for voluntary motions in the pyramids, in their inferior decussation, and in their continuations with the lateral and anterior columns. But it appears from certain pathological facts (marked atrophy of the pyramids *without* any motor affection during life), that they are not the only conductors for voluntary impulses, and are not necessary under all circumstances.

This function of chief motor conductor from the brain to the spinal cord has been assigned to the pyramidal tracts chiefly on the ground of modern anatomical and clinical discoveries. From the latest physiological experiments no binding conclusions can be drawn, and the older experiments would rather lead to an opposite view. The results, too, obtained by the most eminent physiologists are directly contradictory; one saw no particular muscular contractions on irritation, while another observed convulsions; and again after section one saw no paralysis, and the other saw extensive paralysis. According to Schiff the pyramids are not motor conductors, whilst section of funiculi siliquæ and laterales produces motor paralysis. Section of the lateral columns (between the first and fourth cervical nerves in animals) stops the respiratory movements of the same side. The uncertainty and contradiction we meet in the experimental results are easily explained by the difficulty in operating on the anterior surface of the medulla, and the probable variation in the course of the voluntary impulses.

Nothing is definitely known of any *motor conduction in the gray substance*, except what can be determined regarding the nuclei of the nerves and their roots.

The inferior decussation gives a perfectly satisfactory explanation of the *decussation of the motor conducting tracts* on a level with the medulla, a fact which has been proved by numerous experiments and still more numerous clinical facts. Cases that have been observed in which there was no decussation are to be explained by the non-decussation of the pyramido-anterior tracts, and partly, too, by the striking variations the occurrence of which Flechsig has so clearly proved. The pyramido-anterior tracts, in fact, may not decussate before they reach the cord (in the anterior commissure); it is further possible that their decussation has taken place so far forward as the pons, namely in the raphe; thirdly, there is a possibility of their undergoing no decussation whatsoever; but this remains to be proved. There are certainly enough tracts in the raphe and the pons above the motor pyramidal decussation which would all allow of a decussation of motor impulses.

Longitudinal section of the decussation of the pyramids does not produce a complete paralysis of the animal's body; this fact, too, argues against a total decussation, but of course we cannot directly transfer the results to the human subject. Schiff's experiments seem to prove that the decussation of the tracts which supply the muscles of the trunk, the lower and upper extremities, occurs at successively higher points. Schiff's assumption of a repeated ("return") decussation in some tracts requires fresh confirmation.

Sensory Conduction.

The tracts which conduct sensory impulses in the medulla are still quite undefined. Even in the spinal cord we cannot trace the chief tracts with any great certainty, and in the medulla this uncertainty is very considerably increased.

On this point, too, the physiologists are completely at variance. According to Schiff the tracts of the posterior columns, which convey painful impressions, push more and more outwards, and finally come to lie as if in direct continuation of the lateral columns. The floor of the fourth ventricle, he asserts, and likewise the funiculi pyramidum and siliquæ, do not react on irritation. Vulpian, on the contrary, declares that the corpora restiformia and the funiculi graciles et cuneati, and also the floor of the fourth ventricle, are all equally sensitive when irritated. The results of section are quite as contradictory, and can in no instance be called unambiguous or convincing.

It is to be conjectured that the pedunculi cerebelli convey but few sensory impressions, and that this function belongs rather to the central gray substance (formatio reticularis), which conveys them in all directions, so that partial lesions of these tracts never produce any recognizable anæsthesia—of course the tracts which have decussated in the spinal cord continue their further course unaltered—*i. e.*, the tracts belonging to the right half of the trunk are situated in the left half of the medulla, and *vice versa.* But this decussation is by no means a total one, as experiments show us, probably on account of the power the gray substance has of conducting in every direction. If one side of the medulla is cut through, one side of the face is deprived of sensation ; this is sometimes the same as, and sometimes the opposite side to, the section, a matter which seems to depend on the position and depth of the section.

Thus our knowledge of the general situation of the sensory paths amounts to nothing more than a series of conjectures. Of course we know nothing whatsoever of the special tracts for tactile, temperature, and pain impressions.

The functions of the *medulla in the capacity of central organ* are a little more thoroughly investigated and better known. For, in addition to the simple transmission of impressions, the medulla can transfer centripetal irritations to centrifugal tracts ; it can, in fact, bring about *reflex functions*, and these, too, of every kind and with every complication. These appear always to be connected with certain definite, more or less sharply bounded spots, which are for this reason termed *centres* of the several functions. Thus most of the centres are undoubtedly *reflex centres*, and it is only in the case of few that we can assume, with any degree of probability, that they can be thrown into activity without any previous irritation of a centripetal tract, that they act, in other words, as *automatic centres.*

In the first place, the medulla accomplishes *simple or slightly complicated reflex actions*, such as those that pass from the trigeminus in so many ways to the facialis (winking when the conjunctivæ are irritated, wry faces consequent on strong impressions of taste, secretion of saliva when the mucous membrane of the mouth is irritated, sucking motion, when we put a finger into the mouth of a new-born child, etc.). The arc of reflection must be situated in the medulla, and the same may be asserted of the reflex actions between all the other nerves which have their origin in the medulla.

But very much more extensive reflexes take place in the medulla. The so-called *general reflexes*, or the transfer of the reflex irritation to the whole muscular system, cannot be assigned to any other part of the central nervous system. According to Pflueger's laws of reflex action the medulla oblongata is the chief centre of irradiation for all the reflexes of every muscle, and these are never brought into action till the reflex irritation has extended to the bulbus. Ludwig and Owsjannikow have assigned the centre of origin of these general reflexes to the region of the lateral columns in the formatio reticularis.

It is probably the same point from which general convulsions arise in consequence of various direct or reflex irritations. Nothnagel proposes to give it the name *centre of cramps*, and assigns it to the level of the pons in animals. This centre for cramps can be irritated directly (as probably occurs in epilepsy) or reflexly in a great number of ways. According to Nothnagel the reflex irritation is produced with the greatest certainty from two definite points, one on each side of the median line on the floor of the fourth ventricle.

In the human subject the exact situation of this centre is not yet known, but is probably to be sought in the pons portion of the medulla.

Besides these we find in this organ a large number of centres for special processes, some of them, too, extremely important. Some of them effect very complicated reflexes, which display a combination of definite actions to accomplish a definite effect. Actions of this character follow one another in a perfectly regular order, which has its origin in the reflex mechanism and in the performance of certain processes within it.

These centres are partly automatic as well, and their activity persists after all centripetally conducting tracts have been removed, the probable explanation being that they are excited by a certain condition of the blood, and by the changes of the fluid.

The most important and best studied of these centres are probably those which preside over the respiratory mechanism.

The Respiratory Centres.

After Legallois had discovered the centre of respiration, Flourens endeavored to localize it more accurately, and arrived at the conclusion that it was situated in a point not larger than the head of a pin, exactly in the middle line and at the posterior angle of the calamus scriptorius. If this point be destroyed, respiration ceases and life with it (nœud vital). But of late even Flourens himself admits two centres of somewhat larger dimensions.

In point of fact, all more modern investigations point to the existence of *two respiratory centres*, one in each half of the medulla, situated in the superior external portion of each ala cinerea and a little posterior to the exit of the vagus. It is possible that the substance which connects the two may regulate the normal harmony that exists between the respiratory motions of the two sides of the body. Destruction of one centre only affects the respiration of the same side of the body.

Gierke asserts that the "respiratory centre" is nothing but the sum of conducting tracts, which convey the irritations, that set in motion the respiratory reflex from the trigeminus and vagus to the motor-cells of the respiratory nerves. They take the form of a round fasciculus composed of fine nerve-fibres, which runs down on both sides parallel to the median line, and is identical with Krause's respiratory fasciculus mentioned above. So that, in the strict sense of the word, this is not a centre, *i. e.*, a central apparatus composed of ganglion-cells, in which centrifugal and centripetal tracts enter into anatomical and functional connection. Possibly a "respiratory centre" proper may be found in addition to this respiratory fasciculus. Farther down, too, in the spinal cord there may exist similar central structures for respiration (P. Rokitansky), but they certainly play nothing more than a secondary part, and can never come into action independently. The same relation probably exists between these centres as between the vaso-motor centres of the spinal cord and of the medulla.

The centripetal tracts, which come from the periphery and excite or inhibit the respiratory centre, are for the most part situated in the vagus. The centrifugal tracts, which lead from the respiratory centre, run down through the lateral columns to the motor nerves of the different muscles of respiration.

As this is not the place for an accurate acc
ology of respiration, we shall confine oursel
that a certain amount of oxygen must be cont
to maintain the irritability of the respirat
further, the oxygen and carbonic acid conta
must be present in certain fixed proportions
tion of the centres; that the irritation increase
the quantity of carbonic acid exceeds that of
the want of oxygen produces a greater irritati
mulation of carbonic acid. But the respirato
be excited reflexly by a number of different se
as the vagus, trigeminus, and those of the cuti
that the physiologists consider themselves co
separate centres of inspiration and expiration (
four respiratory centres), which appear in certai
an antagonistic action. But neither the anat
mental physiologists have yet been able to loc
accurately.

When the respiratory irritation increases,
affected, till finally general epileptiform con
duced, being the result of an extension of th
the gray substance of the medulla, as far as th

Sneezing, *coughing*, *yawning*, *sobbing*, a
tory (partly in- and partly expiratory) reflex
effected by the respiratory centres of the me
not originate in the same sensory tracts as the
of respiration. All these processes probably h
cial mechanisms, but the anatomical structu
unknown.

Laughing and *weeping* belong to the same
actions, but they are distinguished by their
mimic and secretory reflexes.

Centres for the Cardiac Movem

Although highly probable, it is not certai
dulla possesses a *centre of acceleration* of the a
its position is, of course, unknown.

On the other hand, the excitement of the *inhibitory fibres of the vagus* is generally assigned to a centre in the medulla, though the position of this centre is equally unknown. This "inhibitory centre for the action of the heart" seems also susceptible of automatic and reflex irritation.

Centres of Speech.

The most important *centre for voice*, for the origination of tones, is undoubtedly situated in the bulbar portion of the medulla. For, after the pons has been divided from the bulbus, reflex crying may still be produced. By this process the respiratory centres and the motor tracts of the accessorius (supplying the larynx) are brought into action. Kussmaul (Appendix to Vol. XIV. of this Cycl., p. 650) gives this centre the name of *basal phonic centre*, and considers it indispensable for the construction of articulated sounds. It is situated probably close to the centres of respiration.

Exactly the same may be said of the chief *centre of articulation, i. e.*, the structure which controls the complicated muscular actions necessary to transform simple tones into articulate vowels and consonants. The glossopharyngeus, accessorius, hypoglossus, facialis, and trigeminus, all take part in the production of articulate sounds, undoubtedly a matter of extremely fine, complicated and carefully combined movements.

The anatomical fact that all these nerves and their nuclei lie so close together gives weight to the assumption that the most important centre, at least for the articulation of letters, is situated in the medulla. And this view is further strengthened by numerous pathological facts, which show that diseases of the bulbus, and especially degeneration of the nuclei of its nerves, are particularly destructive to the faculty of speech (progressive bulbar paralysis). There is, however, a slight uncertainty in the case, as these muscles of articulation are always positively paralyzed in bulbar paralysis, so that we are not fully entitled to assume any actual disturbance of co-ordination. It is further quite possible that the real centre of articulation lies higher up in the cerebrum or cerebellum, and that its centrifugal tracts only pass through the medulla, or only set in motion a series of secondary medullary centres. We have, indeed, a parallel for this in the co-ordinate movements of the extremities where this process occurs farther down in the gray substance of the spinal cord. Certainly the centres for the combination of letters to syllables and syllables to words are situated anteriorly to the medulla.

Nothing is known as to the position of this hypothetical centre of articulation in the medulla. Schroeder van der Kolk wrongly assigned it to the olivary bodies, but his view is now rejected by every one. Jaccoud tried to find it in the bulbo-cerebellar commissural system, in the olivo-peduncular region, etc.; but we cannot accept this proposition either. It is more to the point to say that this centre has not yet been discovered.

Centre of Deglutition.

Swallowing is a reflex act that is performed entirely by muscles the nervous supply of which originates in the medulla. It is, in fact, a combined act of the hypoglossus, facialis, glossopharyngeus, vagus, accessorius, and trigeminus. Consequently the centre of deglutition has been assigned to the medulla, an assumption which is well borne out by experimental and clinical facts. But it has not been further localized, nor do we know anything of its connections.

According to Schiff, too, the *centre of vomiting* is situated in the medulla, but nothing further is known about it. Schiff likewise asserts that the *contractions of the abdominal muscles* in defecation are co-ordinated and brought into action by a centre in the medulla.

We can hardly assume a special *reflex centre for the masticatory movements*, as mastication cannot properly be regarded as a reflex action. It is. in fact, only the co-ordination of certain motor impulses in order to accomplish a definite complicated process; the centre of co-ordination which serves this purpose is by no means necessarily situated in the medulla. At the same time, however, it is easy to comprehend that disease of the motor tracts in the medulla which are involved (nucleus of trigeminus, hypoglossus. etc.) might impair the masticatory powers to a great extent.

Vaso-motor Centre.

Ludwig and Thiry demonstrated a centre in the medulla which can throw all the arteries of the body into contraction; and section of the cervical portion of the cord inferiorly to the medulla paralyzes and dilates all the arteries. Later investigations, however (Goltz, Vulpian, Schlesinger, M. Nussbaum, and others), prove beyond doubt that, even if this is the principal one, it is still not the only vaso-motor centre; and further, that other secondary centres exist in the gray substance of the spinal cord, as far down as the lumbar portion. The spinal centres are situated one above the other through the whole cord, and appear dependent, in the first instance, upon the principal medullary centre. When this latter is removed, the spinal centres come into action (but only gradually), and regain their influence over the vessels.

Owsjannikow and Dittmar have defined the position of the vaso-motor centre in the medulla more accurately. They found it, in the rabbit, between the corpora quadrigemina and calamus scriptorius, somewhat removed from the middle line, in the lateral columnar tract of the formatio reticularis. The nerves for vascular contraction which leave it are said to run down through the lateral columns to the anterior nerve-roots (Dittmar).

In addition, centripetal fibres ascend to this centre, which produce a reflex contraction of the vessels; these, too, are said to lie in the lateral columns.

In conclusion, this centre can be excited or depressed by some parts of the cerebrum (irritation of the pedunculi, blushing or pallor resulting from mental impressions).

The latest investigations on the subject of vaso-motor nerves compel us to assume the existence of nerves which directly *dilate* the vessels. For these, too, a centre has been assumed in the medulla.

Nutritive Centres for Certain Nervous Regions.

It seems probable that the gray nuclei of the motor medullary nerves have a nutritive effect upon their nerves and appertaining muscles, very similar to that which the large multipolar cells of the anterior cornua of the cord exercise over the nervo-muscular regions dependent on them. As may be seen in bulbar paralysis, degeneration and destruction of these cells produce granular atrophy of the lingual, labial, faucial, and pharyngeal muscles.

Different clinical and experimental facts have led some to the hypothetical assumption of certain centres in the brain *for the regulation of the temperature of the body.* Should these really exist, we should seek them also in the medulla.

Centres for Certain Secretions.

To Claude Bernard we owe the important discovery that the *secretion of urine*, and certain nutritive processes, can be influenced by irritation of the floor of the fourth ventricle.

If we merely pierce the floor with a needle, half-way between the origins of vagus and acusticus, it suffices to produce a diabetes mellitus. If the needle pierces at another point, closer to the acoustic, it produces a simple polyuria (diabetes insipidus). And, besides this, we can frequently succeed in producing albuminuria by irritation of a point between these two.

In spite of numerous investigations, we are still in uncertainty as to the manner in which this takes place, and as to the mechanism and anatomical structures which bring it about. Claude Bernard conjectured that there was a nervous centre in the medulla which controlled the formation of sugar in the liver. But his view has met with many opponents, and most physiologists now incline to the explanation of this artificial diabetes by vasomotor influences. This view is supported by the fact that irritation of surrounding parts produces only an increased secretion of urine, unaccompanied by any secretion of sugar. But the question has not yet been definitely settled.

Irritation of the anterior portion of the rhomboid sinus, close to the nucleus of the abducens, produces an active *salivation* on the same side as the lesion. The secretion is continuous and abundant. It is, perhaps, produced reflexly by irritation of some branches of the trigeminus. Possibly, however, it is a direct irritation of the secretion, with or without the intervention of vaso-motor processes. Gruetzner's experiments go to prove the existence of a *centre of salivation* in the medulla, which controls both the fibres of the chorda tympani, going to the salivary glands, and the fibres of the sympathetic. And this centre, when directly or reflexly irritated, produces active salivation.

From all the details given in the preceding pages we can at least conclude that a great number of the most important physiological functions are united in the medulla. This very fact compels us to admit the extraordinary entangled and complicated course of the fibres, to which we so frequently drew attention.

III. Diseases of the Medulla Oblongata.

Introduction.

The pathology of the medulla oblongata is still in the first stage of its development. The material for study is relatively scarce, quite unsifted, and in great need of further enlargement; and the only way we can hope to enlarge it is by taking all the modern points of view, and devoting ourselves to a careful clinical study of the cases, and more especially to accurate anatomical investigations.

As might be conjectured from its anatomy, the pathology of the medulla resembles, in some points, that of the cerebrum, and in others that of the spinal cord, so that diseases which are proper to each occur in the medulla. This gives the medullary pathology a very varied character. But, on the other hand, it is simplified, as we can, in many cases, merely refer to the cerebral and spinal diseases, which have in great part been very much better investigated. Some cases—for instance, meningitis of the medulla—we can pass over unmentioned on this account.

As yet we are acquainted with almost
tomatic forms of disease in the medulla. O
most extensive on the subject of *chronic*
paralysis, which had been made known by
gations. Gradually we became acquainted
istic symptoms of an *acute apoplectic bulba*
soon all chronic diseases of the medulla that
ysis were included under the first head, and a
were regarded as apoplectic.

But further observation rendered matters
can already distinguish several different for
classed together under both of these heads, a
in a position now to draw anatomical and c
between these forms.

In the following pages we have endeavo
clinical and pathologico-anatomical distincti
time we are fully conscious that there are
and gaps, and that the material at our dispo
too small, and in particular too inaccurate, t
sure footing on this ground.

The classification we have adopted must
be regarded as a temporary diagram, to conta
the forms of disease with which we are acqu
first discuss the quantitative changes of blood
hemorrhages, and the obstruction of its vess
results of acute and chronic mechanical lesi
and slow compression; thirdly, acute inflam
bus; fourthly, chronic (inflammational) bulba
described by Duchenne; then the other chro
tional diseases of the medulla; and, finally
which occur in its substance.

We must urgently crave the reader's ind
imperfections and defects contained in the foll

1. *Hyperæmia and Hemorrhage of the Medulla Oblongata.— Apoplexia Medullæ Oblongatæ.*

Ollivier (d'Angers), Traité des mal. de la moelle épinière. 3me éd. II. p. 138–167. 1837.—*Mesnet*, Apoplexie du bulbe rhachid. dans sa partie supér. etc. Arch. gén. 1861. Sept. Monit. des Sc. méd. 1861. No. 94.—*Potain*, Paralysie génér. incomplète; polyurie; apoplex. foudroyante; ramollissement ancien, hémorrhagie de la protubérance. Gaz. d. hôp. 1862. No. 93.—*Levier*, Beitr. zur Pathol. der Rückenmarksapoplexie. Dissertation. Bern, 1864.—*Desnos*, Cas d'hémorrhagie de la protub. annul. avec albuminurie, etc. Union méd. 1869. No. 20.—*Wilks*, Labio-glosso-laryngeal Paralysis. Guy's Hosp. Reports. XV. p. 2–17. 1870.—*Hughlings Jackson*, On a Case of Paral. of the Tongue from Hemorrhage in the Medulla Oblong. Lancet, 1872. Nov. 30. p. 770.—*Dutrait*, Hémorrh. sous le plancher du 4 ventricle; albuminurie, glycosurie. Lyon méd. 1875. No. 45.—*C. Browne*, Hemorrh. into the Pons Varolii. Lancet, 1875. Feb. 6.—*Leyden*, Klinik der Rückenmarkskrankheiten. II. p. 63. 1875.—*M. Rosenthal*, Traité des maladies du syst. nerveux. p. 249. 1878.—*Lichtheim.* Ueber apoplektiforme Bulbärparalyse, etc. Deutsches Arch. f. klin. Med. XVIII. 1876.

Pathogenesis and Etiology.

Hyperæmia strictly limited to the medulla must be an extremely rare occurrence, and there is reasonable doubt as to whether such a case has ever been diagnosed with certainty, and a real pathological change ascertained. We are not justified here in adducing the capillary dilatations, which are so frequently found in the medulla, and which are always followed by hyperæmia.

In the great majority of cases hyperæmia of the medulla is nothing more than *part of a general hyperæmia of the brain or spinal cord*, for the medulla is most intimately connected with these in regard to its vascular supply. So that in every case of general cerebral hyperæmia, whether primary or secondary (eclampsia, tetanus, mental and meningitic diseases), the medulla will be found in the same condition.

Naturally the same etiological and pathogenetic conditions, as those that were given at length in Volume XII., when treating of cerebral hyperæmia, retain their force in the medulla. We have partly to deal with active inflammational hyperæmia, resulting from hypertrophy and increased action of the heart, or from vaso-motor disturbances, from collateral congestive inflammation, and from certain poisons, exposure to the sun, or excessive mental activity, etc. But, on the other hand, it may be the result of passive stagnation consequent on cardiac and pulmonary disease—for instance, hindrance to the current in the jugular veins, or violent expiratory exertions (coughing, straining, vomiting, etc.).

We need only say here that all these matters are to be regarded as paving the way for hemorrhage in the medulla, and under certain conditions they actually produce it themselves.

All that Nothnagel has said, in his exhaustive and lucid treatise on cerebral hemorrhage, is equally applicable to *rupture in the medulla*. In regard to hemorrhage the medulla is more closely related to the brain than to the spinal cord, *i. e.*, hemorrhage is much more frequent in the medulla than in the cord; and further, the frequency is greater in the cerebral than in the spinal end of the medulla, so that the great majority of apoplexies are found in the pons division. This fact alone renders it difficult to draw a sharp line between hemorrhage in the pons and in the medulla.

On the whole, rupture of a vessel is a rare occurrence in the medulla. As regards its pathogenesis we may pass it over with a few words, and refer the reader for details to Nothnagel's treatise.

Undoubtedly the most important pathogenetic principle is *disease of the vessels* (miliary aneurisms, such as Heschl describes in the pons, atheroma, fatty degeneration, capillary dilatation, resulting from processes of softening so clearly demonstrated by Gerhardt[1]). We find extremely frequent cases of atheroma and aneurisms of the basilar artery accompanied by hemorrhage from its small branches in medulla and pons.

Increase of arterial tension plays a prominent part in connection with these diseases of the vessels themselves; but even without the latter the rise of the blood-pressure alone can often produce hemorrhage. The same result is seldom produced by an *increased tension in the veins* from stagnation of their blood.

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[1] Jenaische Zeitschrift f. Med. und Naturw. I. 1864.

Under the head of Hemorrhage from Excessive Tension may be brought the cases caused by cardiac palpitation, by great excitement, fits of passion, physical overexertion, and by excessive indulgence in alcohol.

Some diseases of the surrounding parts, which not infrequently lead to hemorrhage of varying extent, seem to act in two ways, *i. e.*, by impairing the walls of the vessels and by raising the collateral blood-pressure. To this class belong caries of the cervical vertebræ, purulent basilar meningitis, and tumors in or around the medulla, etc.

In addition to this, hemorrhage, in greater or smaller degree, is frequently produced traumatically through injuries to the skull and back of the neck, though we can often recognize no direct, palpable lesion in these parts. Westphal produced capillary hemorrhage in the medulla of the guinea-pig by light blows of a hammer on the head. We can imagine something analogous to this occurring to the human subject.

In conclusion it may be remarked, that in extensive cerebral hemorrhage, which breaks through into the ventricles, or in extended rupture in the meninges (*e. g.*, the rupture of an aneurism), the fourth ventricle is often filled with blood, and to this may be attributed some of the symptoms that occur in such cases.

Pathological Anatomy.

As *hyperæmia of the medulla* is characterized by the same anatomical appearances as cerebral hyperæmia in general, we need give no special description of it.

Nor can we draw the slightest distinction between the *apoplectic clots in the medulla* and those in the brain, either as regards their appearance, the changes they go through, or the condition of the surrounding parts. Fresh hemorrhage looks like a dark-red coagulum, and is often half fluid towards its centre. This very soon changes into a semi-solid, chocolate-colored mass, which gradually assumes a lighter color, and is partly absorbed. The last stage consists either in a rusty, contracted scar, or in a small cyst, with a wall of dense cicatricial tissue, with fine threads of connective tissue and filled with a cloudy, yellow fluid. In du the development of a secondary degenerat either unilateral or bilateral, according to rhage; and this degeneration we can trace down the spinal cord.

The *size* of these ruptures is usually very i *shape* is roundish, or resembles an olive or b it is quite irregular. Near the median line small triangular spots of hemorrhage, with forwards, and the base towards the floor of t thus corresponding pretty closely in extent w a median bulbar artery (hemorrhagic infarct ? *situation*, these ruptures can occur at any medulla. If they occur near the floor of t they have a tendency to pierce through it, coagulum of greater or less dimensions in t dom meet with more than a single rupture there are several together. Ollivier describes to six hemorrhagic points in the medulla.

In the medulla alone these effusions of b to any great size, though they are frequentl the pons is simultaneously attacked. In case we often see undefined cystic masses, as large egg, running through the pons and medull into the fourth ventricle, and filling it with a

Capillary hemorrhage occurs, too, occas dulla, in the same characteristic form as in with just the same accompanying or causal ci

Symptoms.

Very little is known of the symptoms of *hyperæmia in the medulla.* It is perhaps some of the symptoms of general cerebral hy slow pulse, vomiting, general convulsions, speech, etc.) to congestion of the medulla, bu thetical. The same, too, may be said of t certain initial symptoms of acute bulbar disease (pains in the head and back of the neck, spasms in the face and tongue, formication, etc.) are to be attributed to hyperæmia in the medulla. This assumption is quite arbitrary, and no proofs have been adduced for it.

In the case of *hyperæmia from stagnation*, we possess some experimental investigations made by Landois, Hermann, and Escher. These prove that retardation of the pulse and dyspnœa (irritation of the vagus and respiratory centres), likewise contraction of the vessels (irritation of the vascular centre), and, finally, general convulsions (irritation of the cramp centre), all result from intense venous stagnation. All these symptoms, without doubt, originate in the medulla, and we are forced to believe that something similar takes place occasionally in the human subject.

Our knowledge of the *symptoms of hemorrhage in the medulla* is more extensive, and rests upon a greater number of observations.

Hemorrhage in this organ, even of inconsiderable extent, is always to be regarded as extremely dangerous, commencing generally with the most alarming symptoms, and not infrequently causing instant death.

It is, in fact, these tempestuous apoplexies (apoplexie foudroyante), from which death results in a few minutes or hours, accompanied by signs of failure of respiration or circulation, that belong usually to the medulla or pons. The patients fall down with a cry or in epileptiform convulsions, and die instantaneously.

Further, we know that large effusions of blood into the hemispheres and ganglia of the base, if they reach the fourth ventricle, and irritate and compress the medulla, very quickly produce death, accompanied by symptoms of vomiting and convulsions, along with deep coma and complete general paralysis.

But slight hemorrhage in the medulla, too, generally produces immediately symptoms of an alarming and very threatening nature—symptoms which come more quickly and are more grave the nearer the effusion is to the centres of respiration, or when it affects these directly. In the latter case instant death may ensue, and, consequently, medical men are seldom called in for consultation or to treat the case.

Our principal interest is consequently att
in which the patient lives on for at least som
in which a partial recovery takes place, and
a somewhat longer existence, though one
materially impaired.

In such cases the patients utter a loud
by buzzing in the ears, dizziness, sudden hea
convulsive spasms of the body, and then foll
general, an apoplectic attack—*i. e.*, they fall
and sink into a more or less deep coma.
paralyses characterize this, as well as all oth
we may localize them often in the medulla
from their extent and combination, and, on t
their almost unexceptional complication with
characteristic symptoms, the origin of which
with a certain amount of probability, to the

The motor paralysis assumes very variou
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upper: at other times it takes a hemiplegi
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most cases all four extremities are either com
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Accompanying this paralysis of the ext
find some of the bulbar nerves more or le
alyzed, usually the hypoglossus, accessoriu
geminus: sometimes, too, the nerves of the o
ses also occur on one or both sides. If we
(an effusion into one side of the medulla), the
symptom is that the paralysis of the bulbar
injury to its nuclei and root-fascicles, occurs
the hemorrhage, while the hemiplegia of the e
the *opposite* side of the body, owing to t
takes place at a point inferior to the lesion
the characteristics of a hemiplegia alternans,
paralyzed on one side, and the facial muscl

this applies as well to the tongue, the muscles of mastication, and to the abducens) paralyzed on the opposite side of the body. The above-mentioned nerves are affected in greater or less number, according to the location of the hemorrhage.

The *paralysis of sensation* follows the same rules as that of motion, only that it is on the whole not so well developed. In severe cases, when all four extremities are paralyzed, it is generally impossible to ascertain anything about the condition of sensation on account of the coma in which the patients lie. If it is a case of motor hemiplegia, we may assume that the sensation decussates as well; but still, owing to the peculiar course of the sensory fibres in the medulla, we can hardly expect a sharply-defined anæsthesia. This, of course, depends altogether upon the extent of the effusion in transverse section. It would be extremely interesting to investigate accurately the condition of sensation on both sides of the head and face in such cases of apoplectic hemiplegia. For *a priori* we should anticipate an alteration in the conditions dominant here, such as a simultaneous, a decussating, or even a bilateral anæsthesia, always provided the so-called ascending root of the trigeminus contains the sensory tract for the same side of the head and face.

Respiratory disturbances, as they are most threatening to the life of the patient, may be regarded as the most important of the symptoms produced by a bulbar lesion, and they are at the same time very characteristic of such lesion. If fatal asphyxia does not at once ensue, still the respiration is always greatly impaired; it becomes irregular, stertorous, often intermittent, and accompanied with the greatest dyspnœa; the Cheyne-Stokes phenomenon is frequently observed; the respiratory process then grows more and more encumbered and paralytic, till at last death results from asphyxia. It is only in comparatively mild cases that the respiration can recover, or is not at all affected. But even in cases which commence as hemiplegia, the hemorrhage generally extends and causes further mischief.

Alterations in the action of the heart are generally less prominent. But in every severe case we unfailingly meet with an enormously rapid pulse, which is frequently irregular and intermittent, and the paralysis of the cardiac action runs a parallel course with the increasing asphyxia. In the cases that have hitherto been described, very little is noted of *vasomotor changes*, unilateral or bilateral rise of temperature in the skin, such as we might expect with certainty, especially in the period immediately succeeding the hemorrhage.

Great importance attaches to the *epileptiform convulsions* which accompany hemorrhage in the pons and medulla. They are often among the earliest symptoms, but they may be repeated in the first few days, and even later, assuming the most violent character. A *tonic rigor* has been often described in the extremities, of variable duration and frequent recurrence.

Disturbances of speech and deglutition, and unilateral and bilateral *paralysis of the soft palate* result naturally from the participation of the bulbar nerves in the paralysis. The same may be said of *aural troubles*, such as deafness and buzzing in the ears, which will probably be more frequently detected now that our attention has been directed to them. Vomiting of frequent recurrence, and a continuous troublesome singultus, which have often been observed, may be explained in the same natural way by an irritation of their centres in the medulla.

Potain, in a case recorded several years ago, describes the occurrence of *polyuria*. Dutrait found both *sugar* and *albumen* in the urine, but his case proves nothing, as both these substances were found before the hemorrhage as well as after. Both Mader and Desnos confirmed the presence of albumen in the urine in the case of an acute hemorrhage, where the kidneys were found quite intact at the autopsy.

In a case where hemorrhage occurred in the pons portion of the medulla, I observed a considerable *rise in the temperature of the body during the agony*. Leyden and others record similar instances, and further it agrees with well known experimental facts.

In cases where life lasts long enough, we can test the electric reaction of the paralyzed nerves and muscles. It probably remains quite normal for some time in the extremities, though it is stated that the irritability of the paralyzed nerves which come directly from the bulbus soon disappears. Leyden has confirmed the latter point, at least as regards the faradic current.

Arguing from analogy, it seems highly probable that when the nervous nuclei of the bulbus, or the nerve-roots that leave them, are destroyed by hemorrhage, the peripheral nerves dependent on them must lose their irritability, and that the muscles supplied by these nerves must give a reaction of degeneration. This will be easily decided by future investigations.

The *course* of the disease is threefold. It is either at once fatal, death being produced by paralysis of the respiratory centres with all the symptoms of *apoplexie foudroyante.*

Or secondly, death does not occur for some hours or days, while the patient lies continuously in a state of more or less intense unconsciousness and is paralyzed to a varying extent, breathing stertorously, and with a quick pulse, sometimes, too, with a high temperature.

In the third, and probably most seldom course, life is maintained for a more considerable time. This only occurs when the hemorrhage is relatively insignificant. The patient gradually recovers his consciousness, some of the paralytic and other symptoms disappear, and nothing remains but hemiplegia or partial paraplegia, and more or less difficulty in articulation and deglutition. Apart from the fact that the actual intellectual region is absolutely unaffected, and apart from any remnants of bulbar paralyses, these cases, in their further course, their termination, and the formation of secondary contractures, etc., exactly resemble those cases in which hemiplegia and other forms of paralysis have been produced by some hemorrhage in the brain anterior to the medulla (in the thalamus opticus, the corpus striatum, the nucleus lentiformis, the capsula, etc.).

* * *

Very little is known of the symptoms of *small capillary hemorrhage* in the medulla; but they are probably similar to those produced by emboli in the smallest arteries of the bulbus, and can scarcely be detected accurately unless occurring on a large scale. There is no evidence as to whether epilepsy can result from this capillary hemorrhage, as Westphal[1] observed it in the guinea-pig after striking it on the head.

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[1] Berl. klin. Wochenschr. 1871. No. 38.

Diagnosis.

It is hardly possible that we shall ever be called upon to make a special diagnosis of hyperæmia of the medulla oblongata.

We can apply the general symptoms of cerebral hemorrhage to the diagnosis of *hemorrhage in the medulla*, so that we need only refer to the chapter on this subject in Vol. XII., and to the description given above. In many cases it is very difficult to distinguish between hemorrhage and embolism. We shall treat of the important points for this differential diagnosis in the next chapter.

In order to make a special diagnosis of a hemorrhagic lesion in the medulla and pons, we must take account of the following: In severe cases loss of consciousness, epileptiform convulsions, and sudden death are sufficiently characteristic to establish the diagnosis. In less rapid cases we may mention the following symptoms as indicative of a similarly situated lesion. Commencement with general epileptiform convulsions, vomiting, singultus, more or less threatening respiratory disorders (intermission, dyspnœa, Cheyne-Stökes' phenomenon), dysphagia, disorders of speech, paralysis of the tongue and soft palate, of the inferior branches of the facialis, and of the abducens, etc.; albumen and sugar in the urine, and final rise of temperature; the extension of paralysis to all four extremities, but particularly the unequal degree of paralysis in the extremities of the one side, and the face and tongue on the other side (paralysis alternans); we might perhaps, too, adduce the cessation of all reflexes in the territory of the paralyzed bulbar nerves.

There are some further isolated symptoms which might make our diagnosis more accurate, but they need some additional confirmation. For instance, we might take the alternating paralysis of the facialis (and perhaps, too, of the masticatory muscles and the abducens) as indicative of hemorrhage in the pons portion of the medulla. But we must take account of several other symptoms according to the extent and exact position of the clot in the pons. As regards these we refer the reader for details to Nothnagel's article in Volume XII.

We may conclude that the lesion is limited to the anterior half of the floor of the fourth ventricle, when we see paralysis of the abducens, facialis, and trigeminus, with aural disorders, and sugar and albumen in the urine, etc.

Hemorrhage in the posterior portion of the rhomboid sinus produces paralysis of the hypoglossus, facialis and trigeminus, and of the accessorius and vagus, accompanied by grave respiratory disorders, and usually by paralyzed extremities; it is a symptom probably of some importance when these latter alternate with the paralysis of the tongue. Alternate paralysis of an upper and lower extremity probably indicates that the lesion is located in the centre of the decussation of the pyramids. In a case described by Hughlings Jackson, a small hemorrhage, which had occurred some considerable time before, immediately bordering upon the left olivary body, is made responsible for a sudden complete paraplegia of the tongue. Whether this is justifiable it would be hard to decide, since there were many other clots found.

Prognosis.

The prognosis is generally very unfavorable; in the great majority of cases, especially when the hemorrhage is of at all large dimensions, it is absolutely fatal. There is only hope of life in cases of very limited hemorrhage, or when the location is very favorable, especially when it is far removed from the respiratory centres. The patient's condition may then improve gradually, and partially recover. As regards details, the prognosis in such cases is the same as in other cerebral hemorrhage.

Capillary hemorrhage, if we could diagnose it, would probably allow of a more favorable prognosis. Still, we should not be sanguine, as the cause—namely, vascular disease—remains to work more mischief.

Treatment.

The rules of treatment for both hyperæmia and hemorrhage in the medulla are exactly the same as those for similar disorders in other parts of the brain. To avoid repetition we shall merely

Pathogenesis and Etiology.

What has been said of hyperæmia can be repeated of *anæmia of the medulla.* It probably occurs seldom or never alone, unless we take account of local compression, in which case anæmia only plays a very secondary part. It is generally a mere component of general anæmia of the whole body, or at least of the brain and spinal cord. We have not to concern ourselves with further details of this, but refer the reader to the article on "Anæmia of the Brain." No doubt some of the symptoms which we see in general anæmia are referable to a similar condition of the medulla.

Thrombosis and embolism of the vessels supplying the medulla are not extremely rare. They are the commonest cause of anæmia of the bulbus; certainly they produce at once the most intense anæmic condition, so that, unless the circulation is quickly compensated, the part at once undergoes necrotic softening.

The *thrombus* or *embolus* occurs almost without exception in the *vertebral and basilar arteries*, and is very seldom confined to the smaller branches of these. Latterly this subject has received much attention; and the result has been that a number of the cases which were classed together as "acute, apoplectic bulbar paralysis" (a term first used by Hérard) were produced by emboli and thrombi in these arteries. Duret, in his monograph on the arteries of the medulla (1873), mentioned all that was important to know; Hallopeau went over the same ground in his thesis (1875), while Lichtheim has given us an excellent treatise (1876), containing everything known of the subject up to the date. However, we are in possession of very few cases which have been under accurate clinical observation, and afterwards received a post-mortem examination. Lichtheim has collected a great many observations, though without the confirmation of an autopsy.

What has been said of thrombosis and embolism in the cerebral arteries in general, applies equally well to these processes in the medulla. The sources of the emboli and the causes of thrombosis are in every respect similar. The basilar artery is frequently atheromatous and contains aneurisms, and it is often infected by syphilis, so that it constantly produces disorders in the circulation of the pons and bulbus by means of thrombosis and contractions in its lumen, though these are occasionally confined at first to a few of the branches it gives off. Thrombi form, too, very easily and often in the vertebral arteries: and in these likewise we often meet with emboli, more especially in the art. vertebralis sinistra on account of its favorable position, given off so directly as it is from the subclavian.

The emboli in these vessels are just as numerous and large as in others, they undergo the same metamorphoses, and disintegrate in just the same way, and are then carried on by the blood. The secondary thrombi and the development of the primary thrombi, till they finally close the lumen of the vessel, are likewise indistinguishable from the process in any other vessels. So we need not enter into any description of embolism and thrombosis, but the process must be borne in mind in order to get the significance of, and enable us to comprehend, the different symptoms.

Pathological Anatomy.

We shall not enter into a description of thrombosis and embolism in the basilar and vertebral arteries, as this can be found in any text-book on pathological anatomy. And besides, in the cases which have been published, the macroscopic anatomical appearances are usually treated of with great accuracy, whilst the consecutive changes in the pons and medulla have been very imperfectly studied.

We shall confine ourselves here to mentioning the facts that either or both vertebral arteries may be completely obliterated; that further, the thrombosis may extend from them into the basilar; thirdly, that the lumen of the basilar may be obstructed either from one end to the other, or for a short distance in its anterior, median, or posterior division, according to which, of course, the symptoms vary. Finally, we may state that the branches given off from the main arteries, partially or wholly, any or all, may be affected by the thrombosis.

The *effect produced* in the medulla is of course greater, the quicker the obstruction is developed and the greater its extent. Almost unexceptionally we meet with grave disorders in the circulation, as most of the nutritive arteries in this region are terminal. The immediate result is, of course, *intense anæmia of the affected vascular territory*. According to the conditions which govern the further development of similar lesions in other parts, the second stage consists either in an *engorgement accompanied by effusion of blood* (hemorrhagic infarct), which leads to red, and afterwards to yellow and white softening, or we have nothing but *simple anæmic yellow and white softening*. The final stage often consists of the formation of a cavity in the bulbus, varying in size, with walls of soft reticular connective tissue, and delicate threads of the same traversing the interior.

If the obstruction takes place in one of the smaller vessels, we find a number of small cuneiform centres of softening, and in them capillary extravasations; or else we meet with similarly shaped hemorrhagic infarcts, with their apices pointing forward and their bases towards the floor of the fourth ventricle.

We are in possession of very few microscopical investigations on the subject of these softenings in the medulla. Charcot found a number of corpuscles of Gluge (Fettkörnchenzellen) in the early stages, and changes similar to what is observed in anæmic parts of the brain. We owe a more accurately studied case to Willigk, who, ten months after the commencement of the disease, found the basilar artery reduced to a fine thrombotic cord, and thrombi in the art. cerebellares inferiores, whilst the vertebrals were unaffected; the pons was atrophied and sclerotic where it touched the fourth ventricle; both pyramids and olivary bodies atrophied. The anterior portion of the pyramids was sclerosed, and from here down Willigk could follow the usual descending secondary degeneration into the lateral and anterior columns of the spinal cord. He found a number of sclerotic vessels filled with detritus and coarse granular substance in the degenerated parts of the pons and medulla, and between these were other vessels which were dilated and admitted of circulation, but were surrounded by lymph-cells, fat and pigment. Everywhere the connective tissue had increased, and the nerves had nothing but disintegrated sheaths and atrophied axis-cylinders. The ganglion-cells were manifestly altered, especially those that lay near the thrombotic vessels; some had undergone granular disintegration and pigmentary degeneration, others were in the last stage of sclerosis and had lost all their processes; the number, too, of ganglion-cells in some parts was greatly diminished, as, for instance, in the anterior portion of the rhomboid sinus, but not so much in the hypoglossal nucleus. An important fact was the marked

average atrophy of the cells in the olivary bodies, whic
diminished in size, while their fibres remained compara
condition reigned in the nuclei of the pyramids. Some o
had atrophied and degenerated, more particularly the ab
of the basilar in this case does not seem to have taken plac
all at once; probably there were several subsequent emb
for the absence of extensive necrosis and softening. Ei
fatty degeneration and dilatation of the vessels, and a nu
Gluge; further, the nerve-fibres and ganglion-cells had d
nective tissue resolved into fat.

Symptoms.

We shall confine ourselves to the sympto
struction in the arteries of the medulla, dr
that seems advantageous, between those tha
main arteries and those in the *small branch*
If *one or both vertebral arteries*, or if *the b*
(thrombosis or embolism), we usually observe
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palate and tongue, loss of speech and the po
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These symptoms are sometimes accompanied
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but it may also be more or less affected.

This acutely developed disease is not of a
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if death from respiratory paralysis did not at once ensue. On the contrary, the patients after some time generally recover to a certain degree. That is to say, the paralysis disappears in part, the extremities contract, the tendon reflexes are more active, etc., as in other cerebral disseminated paralyses, and the patients can remain alive in this condition for a relatively long period.

Duret's thorough investigations of the circulation in the medulla have provided us with means of forming a judgment as to the exact region which is principally affected by obstruction of the different vessels, so that we can draw conclusions as to the location of the thrombosis, from observation of the functions that are disordered. The following is a condensed statement of Duret's results: The important gray nuclei of the bulbus are supplied on the one hand by the arteries of the nerve-roots, which spring from the vertebrals, the basilar and its branches; but they derive by far the greater supply of blood from the median bulbar arteries, which pierce through the anterior fissure into the raphe, and thence to the nuclei. Thus the nuclei of the hypoglossus and accessorius are supplied by branches from the spinalis anterior and vertebralis, and in a very minor degree, too, form the cerebellaris inferior. The nuclei of the vagus, glossopharyngeus and acusticus derive their blood from the branches given off by the anterior portion of the vertebrals and from their junction; the blood-supply of the nuclei of the facialis, trigeminus, abducens, and the two anterior orbital nerves, comes chiefly from the different branches of the basilar.

from

The pyramids and olivary bodies derive their blood in part through small branches coming directly off from the vertebrals, and partly from the spinalis anterior. The pedunculi cerebelli, and also the lateral and posterior portions of the medulla, are supplied by the inferior cerebellar and posterior spinal arteries. The floor of the fourth ventricle receives terminal branches from nearly all the arteries of the bulbus and pons, and in addition to these some vessels from the inferior cerebral artery and from the choroid plexus.

If this vascular distribution were perfectly constant, we could easily make a synthetical estimate of the results that would be produced by the obstruction of any vessel or any point of the same; and on the other hand, we should be able to deduce an exact diagnosis of the location of the lesion from the symptoms presented to us. But the manifold varieties in the points at which these vessels are given off, and in their course, as well as the various possibilities of a collateral circulation, all tend to diminish the success of our efforts in this direction.

We must pay special attention to the unilateral or bilateral origin of the spinalis anterior, and as to whether it unites to form a common trunk or not; further, as to whether the cerebellaris inferior is given off at an anterior or posterior point, etc. With such anomalies a lesion located in exactly the same point of the same vessel may produce in different cases very dissimilar symptoms.

Notwithstanding attempts have been made to localize the thrombus from the symptoms in different cases, and the post-mortem examination has in some of them given results which agreed very well with what was observed during life, still, the diagnostic rules we shall now give can lay no claim to absolute constancy; they are at most approximately true.

Obstruction of the basilar artery, as a rule, produces bilateral symptoms, paralysis of all four extremities, and of both sides of the face, etc.; but indubitably the most important symptom is the cessation of the functions of the vagus and glossopharyngeal nuclei, severe respiratory disorders, dyspnœa, cyanosis, and usually a rapid death from asphyxia. A rapid and complete obstruction of the basilar generally produces instant death. Besides, consciousness is lost in every case, the patients falling down insensible and sinking into deep coma. If life does last for a few hours, we may observe a complete paralysis of all four extremities, caused undoubtedly by anæmia of the pyramidal tracts. But in these cases, too, life soon terminates in asphyxia, the respiration becoming very rapid. Tirard describes cases of 75 to 105 respirations in the minute.

If the obstruction only extends to small portions of the basilar, or if the thrombosis is merely attached to the wall of the artery and only cuts off the blood from a few branches, the symptoms are often less threatening, and individual bulbar nerves are paralyzed, accompanied by weakness or paralysis of some or all of the extremities. In such cases we see the different orbital muscles paralyzed, and those supplied by the facialis, trigeminus, etc. The respiration may then remain uninterrupted, provided circulation continues in the posterior portion of the basilar and in the vertebral arteries.

Obstructions in both vertebrals simultaneously produce exactly the same effects as thrombosis of the basilar artery, namely, complete paralysis of the extremities and the tongue, difficulty in swallowing, aphonia, and intense dyspnœa, quickly followed by death from asphyxia. The life of the patient is not in such immediate danger if the thrombosis is developed slowly enough to allow time for the completion of a slight collateral circulation. Joffroy attaches some importance to the lock-jaw that has been observed in such cases. We are inclined to agree with Lichtheim, who ascribes a case of Marusky's, in which this symptom was observed, to embolism rather than to extravasation.

Obliteration of one vertebral artery produces symptoms which are to a certain degree hemiplegic. The lesion is more frequent in the left artery, from which, too, the spinalis anterior is generally exclusively or in greater part given off. It produces occlusion of the spinalis anterior and cerebellaris inferior. This is the origin of the hemiplegia, which occurs either on the same side as the lesion or on the opposite, a matter that depends on somewhat varying conditions, such as the location of the thrombus, the point of origin of the spinalis anterior, the completeness of the pyramidal decussation, etc. We may also meet with paralysis of the hypoglossus and accessorius, disorders of articulation and deglutition, aphonia, paralysis of the inferior branches of the facialis, partial paralysis and anæsthesia of the soft palate, etc.; but all these symptoms are frequently more prominent on one side of the body. We have thus before us all the symptoms of acute bulbar paralysis, and, in addition, a more or less pronounced hemiplegia or weakness of all four extremities, generally unaccompanied by any loss of consciousness.

We can often recognize the successive occlusion of the different arterial territories quite plainly by the grouping of the symptoms and the order in which they follow one another, which show us how the thrombosis becomes more complete and increases in its extent. This is well illustrated by the cases described by Tirard, Hallopeau, and others.

The larger the vessel occluded and the thrombus occluding it, and the firmer the consistency of the latter, the quicker ensues death. If the circulation can be quickly restored by disintegration or displacement of the thrombus or embolus, or by sufficient collateral branches, we may hope for improvement and partial recovery. Patients of this class may drag out their existence for several years more, though generally retaining severe results of the lesion, such as paralyses, contractures, and difficulties of articulation and deglutition. Lichtheim has described a very good instance of this form.

An *occlusion of the very small branches* in the bulbus can certainly never be diagnosed with certainty from the symptoms it produces. But as these vessels are generally terminal, their obliteration is sure to be accompanied by necrosis of the parts affected, and it only depends on the territory of such an artery whether we get any symptoms of it or not. It is quite possible, for instance, that these occlusions may produce partial paralysis of the tongue, difficulties of articulation and swallowing, unilateral paralysis of the facialis and abducens, respiratory disorders, and perhaps even fits of asthma, etc. But we are seldom or never able to conjecture such lesions.

To this category, however, belongs a case of Voisin's, which Duret quotes. A woman had all the symptoms of a suddenly developed bulbar paralysis, and the post-mortem examination proved the presence of a triangular capillary clot as large as a pea, situated in the middle line and pointing with its apex to the anterior surface of the bulbus, while its base was directed towards the floor of the fourth ventricle. Hallopeau, too, describes an interesting case in which the main lesion consisted in a small point of softening in the bulbus, which coincided exactly with the common nucleus of the facialis and abducens, and had paralyzed these two nerves. An embolus of the vertebralis as well had given rise to hemiplegia on the opposite side of the body, while a continuous incomplete thrombosis of the basilar produced other paralytic symptoms, and finally caused death from asphyxia.

Diagnosis.

The diagnosis of a bulbar embolus or thrombus is very frequently surrounded with many difficulties, though in most cases we can scarcely fail to inform ourselves of the approximate location of the well-marked symptoms. A bulbar paralysis, for instance, with all its characteristic symptoms of difficulty in swallowing and articulation, palato-labio-lingual paralysis, partial paralysis of the masticatory and some of the orbital muscles, with threatening disorders of the respiration and circulation, and accompanied by either hemi- or paraplegia of the extremities, occasionally too by epileptoid convulsions—all this is so characteristic that we can hardly fail to comprehend it, and our attention is at once directed to the pons and medulla. But in fulminant and extremely rapid cases, when the patient sinks into deep coma, and universal paralysis sets in with threatening asphyxia, we can only make a guess as to where the lesion has occurred.

We can generally make a pretty positive diagnosis of an acute, defined lesion in the bulbus, if the symptoms are suddenly developed without any warning; for, even in a case of autochthonous thrombosis, we generally have a sudden attack, probably the moment the vessels are completely occluded. But it is not always easy to determine whether a hemorrhage or an embolism has taken place. The general grounds for the one or other interpretation are, as is well known, not at all absolute (compare Nothnagel, Vol. XII.); nor can we always find any source of an embolus; besides, in old persons thrombosis is just as frequent as hemorrhage, and in young subjects the arteries may be infected by syphilis. These reasons render a positive distinction, in most cases, impossible, unless the special symptoms lead us to diagnose one cause or the other. This is, however, very frequently the case.

We could hardly entertain thoughts of an extravasation, for instance, in Hallopeau's case, where the different symptoms came on one after the other—the same effect as produced by an embolus with continued development of a secondary thrombus.

Epileptoid convulsions and apoplectic attacks are of more frequent occurrence in cases of hemorrhage than in those of embolism and thrombosis; it is only when the whole basilar is completely occluded that we meet with a severe apoplectic attack.

The symptoms of embolism are, in many cases, of a definite character, owing to the regular distribution of the vessels, whilst

those of apoplexy are more dependent on cl
oftener meet with a repetition of exactly th
symptoms in cases of embolus than we do in e
an instance of this repetition, we refer the read
case of obliteration of the vertebral, with sim
paralysis and paralysis of the extremities.

The course of the disease is very frequently
or against a diagnosis of embolism. Striking a
ment, with total disappearance of complete gr
symptoms, seldom occurs in cases of hemorrha

When we have decided on a thrombus or
perhaps only on grounds of probability, we mu
exact localization of the lesion. This will often
we can go upon the basis of the facts stated abo
nosis must always have a certain amount of u
to all the possible varieties of the vessels. Bes
tion of the paralyses, other symptoms may he
diagnosis. Thus, in diagnosing occlusion o
might take account of an unusually full pul
and of Griesinger's symptom (which he deduced
we have never heard of its being practically
general convulsions resulting from energetic co
carotids, just as in Kussmaul and Tenner's e
practical application of this experiment seem
what problematical.

Prognosis.

We need scarcely observe that the cases
thrombosis with which we are now occupied a
character. Whilst the prognosis of sudden and
tion of the basilaris or both vertebrals is almost
we may say of a slowly developing, or at first i
sion of one or more of the large vessels in th
almost unexceptionally ends in death within a s
cannot expect life to last long, except in cases of
obstruction, which happen to affect the least da
where a possibility is offered of a somewhat co

eral circulation. When such cases become chronic they share the same prognosis as thrombotic or embolical softening of other cerebral regions, and are consequently, in their later periods, to be regarded as incurable.

Treatment.

We refer the reader, for the treatment of these cases, to Nothnagel's article. Stimulants and tonics are plainly indicated when the thrombosis is located in the bulbus; but, unfortunately, the diagnosis is so uncertain in many cases that it is difficult to follow out any definite therapeutical course. So that we must be guided by the scanty symptomatic indications, which can be gathered from a careful observation of the individual case.

At a later period electricity may be applied with most success. Markusy derived the most wonderful results from its application in a case which probably belonged to this class.

3. *Injuries and Wounds of the Medulla Oblongata.—Chronic Compression of the Same.*

Ollivier, l. c. 3me éd. I. u. II. *passim.*—*Waters*, Remarkable Case of Lesion of the Medulla Oblong. Medico-Chir. Transact. Vol. XLVI. p. 115. 1863.—*Vix*, Fall von einseit. Verletzung des verl. Marks. Correspondenzbl. d. ärztl. Ver. im Rheinland, 1874. Centralbl. 1875. No. 22.—*Leyden*, Klinik I. und II. 1874–76.—*Hallopeau*, Des paralysies bulbaires. Paris, 1875. p. 117.—*C. Lange*, Ein paar plötzliche Todesfälle. Hosp. Tid. 1870. Virchow-Hirsch. Jahresber. 1870.—*Levrat-Perroton*, Cas de glycosurie déterminée par une tumeur colloïde renfermée dans le 4me ventricule. Thèse. Paris, 1859.—*Solbrig*, Ueber die Verengerung des Eingangs des Wirbelcanals in den mit Epilepsie verbundenen Seelenstörungen. Allgem. Zeitschr. für Psych. Bd. 24. p. 1. 1867—*v. Recklinghausen*, Virch. Arch. Bd. 30. p. 364. 1864.—*Ladame*, Symptomatologie und Diagnostik der Hirngeschwülste. p. 43. 1865.—*Voisin*, Cas de paral. glosso-lab.-lar., sans sclérose du bulbe. Annal. médic. psych. Janv. 1871. Virchow-Hirsch. Jahresber. 1871. Vol. II. p. 59.—*Baelz*, Enchondrom der Schädelbasis. Arch. der Heilkunde. XIII. p. 192. 1872.—*Bouchard*, Compress. de la pyram. antér. gauche par l'apophysie odont.; hémiplégie; convulsions épilept. (quoted in Hallopeau's work, p. 118).—*Bourdon*, Tubercule du cervelet occupant l'éminence vermicul. et comprimant le bulbe; embarras de la parole. ibid. p. 125.—*Garrod* and *Philpot*, Papillomatous Tumor in the Fourth Ven-

tricle of the Brain. Lancet. 1873. March 1.—*Herts*, Fall von Erweichungsherden in der Med. oblong. etc. Deutsches Arch. f. klin. Med. XIII. p. 385. 1874. —*Rotter*, Arthritis deformans der Articul. epistropheo-atlant. etc. ibidem XIII. p. 403. 1874.—*Verron*, Étude sur les tumeurs du 4. ventricule. Thèse. Paris, 1874.—*Hallopeau*, Tumeur dans le cerveau moyen. Gaz. méd. 1874. No. 9.—*Hubrich*, Geschwulst des Kleinhirns, Druck auf die Med. oblong. Arch. für Psych. u. Nerv. V. p. 594. 1875.—*Lansoni*, Caso singolare di paral. bulb. per compress. di un tumore del cerveletto. Raccogl. med. 1876. Aprile. Jahresber. pro. 1876. II. p. 111.—*Morelli*, Affectionen der Med. oblong. Centralbl. 1877. No. 38.—*Griesinger*, Aneurysma d. Basilararterie. Gesammelte Abhandl. I. p. 472 (from the Arch. d. Heilkunde III.).—*Lebert*, Aneurysma d. Vertebrales, Basilaris, etc. Berliner klin. Wochenschr. 1866. No. 28.

Notwithstanding its protected position, the medulla oblongata often suffers from traumatic influences, and is thereby, directly or indirectly, more or less injured and even destroyed. To this category belong *acute mechanical injuries* to this vital organ, which are of the most dangerous character, and consequently have, as a rule, a very short clinical history.

But, besides these, there are other mechanical influences which affect the medulla from without, but quite gradually and increasing by slow degrees. These influences are neoplasmata, disease of the bones, and dislocations, parasites, etc. They all produce a *slowly increasing pressure*, and compress the medulla chronically.

It appears to us quite suitable to discuss the two—the acute and chronic forms of mechanical injury to the medulla—in the same section.

Pathogenesis and Etiology.

Injuries of the medulla may be produced in many different ways: for instance, by a cut or stab, which pierces easiest between the atlas and occiput; or by foreign bodies, such as pistol and rifle bullets, splinters of bone, etc. Such injuries can occur at any point, accompanied or unaccompanied by injuries of the cervical vertebræ and skull. Further, these wounds may be produced by a cut on the head, with or without fracture of the skull, and in many cases by contre-coup. A fall on the head or back of the neck, or a heavy body striking these parts or the face, produces similar results.

Fractures, dislocations and displacements of the two first cervical vertebræ are very important and frequent causes of wounds of the medulla. When the vertebræ are fractured, as happens from gunshot wounds, or from a fall or blow on the head or neck, etc., we always find them more or less displaced; if this is not the case, the medulla may escape uninjured. The most dangerous luxation is that of the first vertebræ, as it is almost always accompanied by backward displacement of the odontoid process; this process is then forced against the anterior surface of the medulla, which it compresses and lacerates, especially when some pathological process has already produced enlargement of the bone, or altered its natural direction of growth. This event, followed by immediate death, can also be produced by simple *dislocation of the odontoid process.* Dislocation can take place when the head is sharply pulled forward, as in hanging, or flexed, the epistropheus slipping from under the transverse ligament. The same effect is produced by any great force acting on the head and neck, inducing a luxation in the occipito-atlantoid or atlanto-epistropheal articulations. A similar result ensues from caries of the first cervical vertebræ, which slackens and destroys the ligaments and the vertebræ themselves, so that quite a trifling mechanical process, such as a sudden turn of the head, etc., produces dislocation.

The causes of *slow compression* of the medulla are very various. If any neoplasma or enlargement (whether it arises in the bone, periosteum, meninges, in the vessels or the surrounding parts of the brain) grows in the direction of the medulla, it may produce slowly increasing compression, and thereby cause material injury to the functions of this organ.

The processes of this class with which we most frequently meet are osseous. *Simple caries* of the occipital bone and two first cervical vertebræ may produce the effect, with its exudations, abscesses, osseous tumefaction, dislocation of the odontoid, etc.

The medulla is often compressed, too, by *abnormalities in the shape and size of the bones.* Solbrig directs attention to one

of these, which is by no means of seldom o
constriction of the entrance to the spinal can
misgrowth of the processus anonymi occipiti
odontoideus, and of the posterior semicircle of
cases of epilepsy he found at the autopsies co
tion, which had caused compression. And
similar cases. An *enlargement of the odontoi*
Hertz describes a case in which this process
larged, with its point directed sharply back
medulla, perhaps the result of rachitis. Thes
seem to have any absolute significance, and a
mere chance; but when combined with certain
influences or some casual effort (physical ove
a heavy load, etc.), they may give rise to gr
Hertz's case, for instance, the overgrowth of
produced a small centre of softening in the
pyramids, corresponding exactly to the point

Arthritis deformans in the articulations
put, atlas, and epistropheus, would seem t
rarer cause of compression. Rotter has latel
in which the shape of the joints was imme
odontoid process enlarged, and the ligaments
the occipital foramen was considerably constr

The medulla is much oftener compressed
from the bony parts and growing into the
Baelz found an enchondroma from the base of
a cystosarcoma of the clivus, and Hallopeau
tumor from the base, and we could enumera
stances of this class.

The same effect is produced by differen
favorably situated on other surrounding s
syphilitic gummata arising on the *dura mat*
the *choroid plexus* (Kussmaul), epitheliomata
(Voisin), etc.

In this class we may further include *aneur*
and vertebral arteries, which are of somewh
rence; they, however, in the majority of c
pons, but sometimes their influence extends

its nerves. They are rendered still more dangerous by the thrombosis, which almost constantly accompanies them.

Lastly, we come to *tumors of the cerebellum*, which, when they are large enough to compress the medulla and are favorably situated, play a very prominent part. Cases of this kind are numerous. Bourdon describes a small tubercle in the vermis cerebelli surrounded by an exudation, which compressed the inferior portion of the rhomboid sinus; Morelli had a case of tubercle on the floor of the fourth ventricle; Hubrich saw a glioma, probably originating in the flocculus, and wedged in between the medulla, cerebellum, and pons, which had compressed the medulla considerably, and pushed its nerves to one side; Lanzoni observed a glioma as large as a hen's egg in the vermis inferior, which covered the fourth ventricle; Voisin found an epithelioma in the pia mater of the cerebellum growing in the direction of the medulla. But we need not multiply cases further.

The action in all these cases is increasing pressure from without upon the medulla, and this pressure must naturally first act upon the nerve-roots given off by the medulla, and then upon the different parts and funiculi in the organ itself. This is what produces the characteristic symptoms of gradually increasing irritation and paralysis. But frequently a long time elapses before we can observe any symptoms, or perhaps only very insignificant ones; and then these cases exhibit suddenly the most acute paralytic symptoms, ending quickly with death. This, of course, leads us to assume an embolus or extravasation, but this occurs by no means constantly; on the contrary, it is almost certain that bulbar myelitis produced by compression, which extends rapidly over a large surface, is the origin of these acute symptoms. In fact, we have an analogous case in the slow compression of the spinal cord. It is the possibility of this myelitis by compression that constitutes the chief danger in a great many cases of mechanical injuries of the medulla oblongata.

Pathological Anatomy.

It is unnecessary to dwell upon the causes which could produce an *acute trauma* in the medulla. A few words, too, will dispose of the anatomical changes which we (when wounded, lacerated, or crushed. The; detail the acute injuries in the spinal cord tion is similar. The location and extent thes very various, and of course dependent on cha

It would also take up too much space t tomical appearances of the causes of *slow c* shall content ourselves with having enumera

The medulla itself may undergo the mo from compression: its shape may be alter located, turned on its axis, or pressed out fl dered unsymmetrical, or may receive a numb various depth and size. Various parts are atr or there may be sharply-defined indentations severely compressed, whilst the other is relati It is quite plain that all this depends entirel and shape of the compressing processes, and accountable for the different symptoms that

Relatively little is known of the histol take place in such cases. We usually find th and generally *softened.* In Hubrich's case t soft and friable. Extravasations in the softe quent occurrence. In the small centre of so describes, he found that the medullary sheat and likewise part of the axis-cylinder; he sa of finely granulated, soft, interstitial tissue, cles of Gluge, and corpora amylacea; the vess dilated, hypertrophied, and covered with g pigment, and it was around them he found cles of Gluge.

The nerve-roots given off from the bulbus or less degenerated and compressed; they a attenuated, and undergo degenerative atroph

Secondary degenerations often arise from run down into the spinal column.

However, in future cases it would be very more accurate investigations on the results medulla from compression.

Symptoms.

If the *injury* to the medulla is a *severe*, *acute* one, the symptoms are generally very simple—the patients collapse as if struck by lightning, and die instantaneously; sometimes they give utterance to a loud cry before falling, or death may be accompanied by a few transitory convulsions. All this results from sudden paralysis of the respiratory centre, and complete interruption of every conducting connection with the spinal cord and the periphery.

This is the ordinary course when the occiput is dislocated towards the atlas, or when the odontoid process is forced from the epistropheus, or in fractures of the anterior cervical vertebræ, or in gunshot and knife wounds of the medulla, and also in a number of other cases.

When the wounds are relatively less severe, or when the medulla is only partially lacerated, life may last for some time; but these cases, too, end suddenly.

Waters describes a case of this nature. The patient received a heavy blow in the face, unaccompanied by fracture of the skull. At first the symptoms were trifling, consisting of a slight feeling of weakness, and deafness on the right side. There was no anæsthesia; the tongue and uvula were slightly oblique; the patient suffered, too, from singultus, and was unable to swallow. The temperature was high on the right side. Sudden death in twenty-four hours. At the autopsy he found both sides of the right pedunculus cerebelli lacerated, the wound extending to the floor of the fourth ventricle. The paralytic symptoms may be explained by laceration of the nuclei, and death probably resulted from paralysis of the respiratory centre.

Vix's case cannot be regarded as anything more than a mere unilateral wound of the upper cervical portion of the spinal cord, which was located in all probability *below* the decussation of the pyramids. No conclusions, however, can be drawn from the case, as the patient survived, and an autopsy was never obtained.

It is certainly highly probable that patients sometimes survive very small injuries to the medulla. But still this is difficult to prove; for, from the symptoms of paralyses of different kinds, troubles of articulation and hearing, epilepsy, slow pulse, diabetes mellitus or insipidus, etc., we can only *conjecture* that the medulla has received some injury.

On the other hand, it is by no means ce
always assume an actual lesion such as hem
or comminution, to explain the symptoms
matic influences bearing upon the neck and s
be difficult to account for all the symptoms
tion, similar to what has been found in the
not alter the question whether they are acc
chronic nutritive troubles. This remains to
is no doubt, however, that our present mate
to allow us definitely to assume the existe
medullæ oblongatæ.

Special importance attaches to a number
in slow compression of the medulla. The
develop very gradually, and may escape obs
is very variable, and they are the results of i
sis of the nerve-roots coming from the bulbu
first place, we have symptoms of *irritatio*
varying intensity in the territory of the trige
face-aches, which often assume a neuralgic
generally unilateral, though sometimes bilat
further belong twitching of the muscles sup
transitory cramps in the tongue and lips;
meet with clonic and permanent contractions
with buzzing in the ears, etc. When the bulb
irritated, we may observe epileptoid convulsi
ness, and singultus.

After these initial symptoms have lasted
the nerves above mentioned show *signs of*
accompanied with *atrophy*, and thus we g
symptoms of a "*bulbar paralysis.*" *Paral*
tics and disorders of respiration and circulati

But the development of the disease does
course; sometimes it *begins more acutely*—
that we are induced to think of an embolus,
similar lesion. Cases of this nature are reco
Bourdon, Bouchard, and others. In point
commencement may be caused by thrombos
the medulla; but in the majority of cases it

idly developing and spreading myelitis from compression, after this latter has reached a certain intensity.

In both cases, if the symptoms are well developed, we have, in the first place, *paralysis of the bulbar nerves and centra*, varying in extent, symmetry, and intensity ; in the second place, the extremities are paralyzed—either a perfect hemiplegia, or, when paraplegia, the symptoms are more marked on one side than on the other. Lastly, we meet with different degrees of *sensory disorders*, both in the face and on the trunk and extremities. But, at the same time, there is no sign of any anterior parts of the brain being complicated, and consciousness is generally quite unimpaired. The optic nerves are never affected, except when the tumor is very large, and so situated as to extend its influence thus far.

It is a most striking fact that in this disease we usually meet with a group of symptoms which are generally taken to indicate bulbar paralysis, and almost all the cases of this nature have been described under the heading of chronic bulbar paralysis. To this class belong paralysis of the tongue and muscles of articulation, partial paralysis of the inferior branches of the facialis (especially as regards the lips) and of the soft palate, troubles of deglutition, increased secretion of saliva, hoarseness and aphonia, disturbances of the respiration and circulation, and occasionally paralysis of the orbital muscles (especially the abducens) and the masticatory muscles, anæsthesia in the territory of the trigeminus, deafness, and singing in the ears; finally, we may meet with vomiting, singultus, epileptic convulsions, signs of vascular paralysis, Stokes' respiration, and a number of other symptoms which do not need mention. Of course, all these symptoms do not occur, by any means, in every case, but only one or more at a time, so that these cases of slow compression assume very various forms. This variability of symptoms is the great characteristic which distinguishes it from "chronic progressive bulbar paralysis." This latter disease, which we shall describe later on, presents unusually constant and sharply defined symptoms.

To illustrate the variability of symptoms in slow compression, we shall give the following summaries of cases on record :

Bouchard's case: *Compression of the left pyramid ;* hemi
with contractures, followed by epileptoid convulsions conf
other lesion found in the brain.

Hallopeau : Compression of both pyramids ; consequent
and later, of both lower extremities, with permanent c
remittent spasms in the members; no anæsthesia; finally,

Hertz : Myelitis from compression in the decussation
paralysis of all four extremities, with spasms and contra
neck too; contracture of the masticatory muscles; speec
sation unaffected; remarkable temporary redness of the fa
and headache.

Rotter : Compression in the region of the inferior decus
caused by the odontoid process ; motor paralysis of the extr
and of the sphincters; epileptoid convulsions; no disord
and deglutition unaffected. (Imperfect record.)

Baelz : Enchondroma of the base of the skull, compressi
and hardly affecting the bulbus itself at all; headache; n
nus; convulsive fits in the lips and tongue; in a later stag
sis, with paralysis and atrophy of the tongue; finally, ana
scalp; extremities very little affected.

Hubrich : A glioma, as large as a walnut, wedged in on
medulla, cerebellum, and pons ; headaches, buzzing in th
troubles of deglutition, and a feeling of fur on the hands
saliva; paralysis of both inferior and of the right superio
sis of the lips or tongue, but speech somewhat affected;
attacks of vertigo and weakness, and finally, loss of conscio
and passing blindness. The patient himself assigned the l
racy, to the point where it was found at the autopsy.

Leyden : A cystosarcoma which had forced its way on th
medulla, cerebellum, and pons ; severe headache; vertigo;
later, general weakness of all four extremities, with violen
paralyzed bladder, and decubitus.

Bourdon : The floor of the fourth ventricle compressed by
the nucleus of the hypoglossus ; speech very much impaire
bulbar functions unaffected; sudden death.

Morelli : Tubercle on the floor of the fourth ventricle ;
paired vision; paralysis of right facialis and right abducen
upper extremity; fibrillar twitchings in the tongue and left

Lanzoni : Glioma on the floor of the fourth ventricle ;
vomit, dyspnœa, unsteady gait, strabismus, tongue to the
ing and swallowing, facialis paralyzed on the left side; no
ities, sensibility unaffected.

Chouppe (in Verron's work, p. 40): *Hypertrophy of cho*
thickness of one centimetre; *pain in the face, frequent vom*

decrease of intelligence, and amblyopia; partial paralysis of the facialis on the right side; sensibility unimpaired; later, delirium; contraction of the extremities of the left side; *neither albumen nor sugar.*

Von Recklinghausen: Hypertrophy of the choroid plexus, unattached in the ventricle; scalp-wound; later, loss of memory; *diabetes mellitus;* phthisis.

Bourdon (in Verron's work, p. 52): *Tumor of the choroid plexus as large as a hazel-nut*, of firm tissue, and like a mulberry; secondary softening of the pyramids; convulsive movements, and tremor in the extremities, tongue, and face, rendering difficult, too, the movements of the eye and of speech; general muscular weakness; normal sensibility; *frontal headache;* decrease of intelligence; no albumen or sugar; finally, continuous convulsive contractions, speech incomprehensible, respiration and deglutition unaffected, mania, vomiting, death.

Garrod (in Verron's work, p. 57): *Papilloma of the choroid plexus*, one and one-half inches long, occupying the whole ventricle, very vascular; wound on the head; *unsteady gait;* ataxy; weak sight, and stagnation in the papilla; *paralysis of right abducens;* strength of muscles almost unaffected, and normal sensibility; *pain in the back of neck;* weakened intelligence; *vomiting; deafness*; difficulty in swallowing; disturbances of respiration and circulation; partial paralysis of the right facialis; finally, coma, general paralysis, and a terminal rise of temperature.

Solbrig's cases: *Contraction of the entrance to the spinal canal;* probably in all the cases a very chronic, slow compression of the medulla; as yet only found in epileptic insane, but probably causally related to epilepsy. But we need more details.

These cases will suffice to give an idea of the great number of different possible symptoms, and at the same time show the difficulties we have to deal with and the points to which we must hold fast in the interpretation of individual cases.

Diagnosis.

The diagnosis of a severe *acute trauma* of the medulla is seldom made during life, and generally only after a *post-mortem* examination; and in the few cases in which life remains unextinguished for a few hours, we must hesitate to make a certain diagnosis on the very ground that death did not follow instantaneously. In the latter case the probability-diagnosis can only be confirmed by a sudden death. Even in lighter cases, where the symptoms remain constant, the most careful estimation of all the circumstances can hardly lead to anything more than a probable conjecture.

The symptoms of *slow compression* are so grouped and succeed one another in such an order, as to allow of a diagnosis in many cases. Thus, when we observe a series of initial symptoms of irritation in the bulbar nerves, sharply confined to certain nervous tracts; and then in the same tracts symptoms of paralysis which, perhaps at an early stage, prove on electrical examination to be peripheral; when we meet with giddiness, violent headache, vomiting, and epileptic convulsions; and when, later, these are followed by the typical symptoms of bulbar paralysis, complicated with paralysis, twitching, and contractures in the extremities; and if these symptoms of paralysis show a disposition to spread rapidly; if they are rather unsymmetrical on the two sides; and if, finally, the sensorium is unaffected—in such a case we may assume a gradually increasing compression from without with considerable certainty.

But it is different when the initial symptoms are absent or very trifling, and we suddenly meet with all the symptoms of an "acute bulbar paralysis" (see next paragraph) simultaneously with the development of a myelitis from compression or the occurrence of hemorrhage or thrombosis. Here it would be impossible to make a certain diagnosis, though perhaps there might be a possibility, by means of more accurate investigation, of finding some previous suspicious symptoms from which we might conjecture that it was not an actual case of so-called primary acute bulbar paralysis, but only a secondary one, originating from compression from without.

The chronic cases are easiest and oftenest confounded with *Duchenne's chronic bulbar paralysis*. In our opinion it is not so very difficult to distinguish between the two forms, provided that we understand Duchenne's disease as a well-characterized and sharply-defined form, and not merely any chronic paralysis of the bulbar nerves and centres whatsoever. We shall discuss this point more in detail in Paragraph 5, under the head of Chronic Progressive Bulbar Paralysis.

Cases of slow compression of the anterior half of the medulla alone, where the pyramids are mainly involved, exhibit a very striking likeness to *spastic spinal paralysis*, so that, when the disease is fully developed, the resemblance is almost complete (compare Hallopeau's case, l. c., p. 121). Still, in most cases it will be possible to draw a distinction, though, of course, it cannot be a reliable one until we become acquainted with the anatomical lesion which causes spastic spinal paralysis. The points of distinction are: the sudden commencement of the paralysis, the fact that it appears first in the upper extremities, and that it is followed later by actual bulbar symptoms, dyspnœa, paralysis of the tongue, difficulty of swallowing, etc.

But, even when we do diagnose slow compression of the medulla, there still remains the difficulty of *determining its cause.* Here we must proceed on general principles. It often happens that we find characteristic symptoms which enable us to say whether it is a case of caries of a cervical vertebra, an aneurism of the basilar artery, a tumor within the skull, or syphilis. Still, too, these frequently fail us.

Prognosis.

Every severe *acute injury* to the medulla causes instant death; and smaller lesions, too, are usually extremely dangerous, because the traumatic inflammation and softening consequent on them often lead rapidly to a fatal termination. We can seldom hope for any long duration of life.

The prognosis of *slow compression* of the medulla is, of course, dependent upon the cause of the compression; but in most cases it may be pronounced absolutely fatal, as most of the processes in this category follow an uninterruptedly progressive course. The prognosis of the quickness or slowness of the disease in producing death depends, on the one hand, upon the rate at which the structure grows, and, on the other hand, upon the point at which the medulla is attacked, and whether the vital centres come at once or only slowly under the influence of compression.

We may form a prognosis not absolutely fatal in cases of simple caries (as restitution is possible), in arthritis deformans, and in syphilitic tumors.

It naturally needs an exceedingly careful estimation of every circumstance and symptom before we can form a prognosis for each individual case.

Treatment.

Little can be said on this head. Acute inju
must be treated in the same way as similar
cord.

Causal treatment is the chief and only m
ises success in slow compression. We can fi
is to be done in cases of caries, syphilis, aneu
We must take a purely symptomatic cours
diagnosis is uncertain, it would be advisable
a number of empiric remedies.

4. *Acute Inflammation of the Medulla Obl*
Bulbi Acuta.—Acute Bulbar Pa

Abercrombie, Abscess of the Medull. Oblong. Diseases o
Cord. p. 108. Edin. 4th ed. 1846.—*Lange*, Om acute
Tidende. 1868. Nos. 51–53. Jahresberichte pro 1868.
scess in der Varolsbrücke, etc. Oesterr. Zeitschr. f. pra
—*Rosenthal*, loc. cit.—*Leyden*, Klin. der Rückenmarks
—*Leyden*, Zwei Fälle acuter Bulbärparalyse. Arch. f.
44. 1876.—*Lichtheim*, Ueber apoplektiforme Bulbär
Archiv f. klin. Med. XVIII. 1876.

It is only in the last few years that this d
its due share of attention. Under the heads
lism, myelitis from compression, and trauma
bulbus, we have, in the preceding paragraph
plectiform bulbar paralysis." But there still
cases of great interest, and which must mo
garded as primary acute myelitis bulbi. Le
who drew our attention to this form, and he
an accurate description of three cases.

The other cases, which are scattered over the literature
well taken into consideration: Abercrombie's case of an
been a tubercle, and must consequently be excluded; Me
abscess, but his case was complicated with basilar menin
are doubtful, and perhaps belong to another form of diseas
to us Leyden's three interesting cases, and we have taken
our description.

We shall bring together under this head the *processes in the medulla oblongata of relatively acute and primary origin*, which may fairly be designated as inflammational. On the other hand, we do not include forms of secondary origin, such as the bulbar processes, which originate in acute ascending myelitis, and usually bring about the fatal termination of acute central myelitis, and ascending poliomyelitis anterior subacuta. Nor can we include the myelitic centres we find in the oblongata in cases of basilar meningitis.

Etiology.

The causes of acute bulbar myelitis are still almost unknown. If we put out of consideration the myelitides ascending from the spinal cord, and also basilar meningitis and emboli as causes of centres of softening, we have nothing further.

In Leyden's first case he could adduce no cause; in his second he assumes an embolus as the probable origin, but no embolus was found; it was preceded by rheumatic arthritis. His third case was a hard drinker, who had suffered greatly from rheumatism, but had no peculiar antecedents.

Pathological Anatomy.

Rather small centres of softening, without definite boundaries, were found in the medulla in all three cases. In two cases nothing abnormal could be seen on the fresh specimens with the naked eye, either in the form, color, or consistence of the medulla. But in the third case, even without the microscope, a centre of extravasation could be plainly seen, dotted with numerous small extravasations. When the specimens were hardened, however, the position and size of the myelitic centres were easily recognized.

An accurate examination gave the following results:

Case I.—Woman, thirty-six years of age. Region of the pyramids and olivary bodies covered with numerous small extravasations; all around the softened substance was of a gray and reddish yellow tinge. These centres were found from above the decussation of the pyramids up to the inferior position of the pons, occupying especially the so-called internal fields of the medulla, and were more numerous on the left side than on the right; on the left side changes had taken place between the roots of the vagus and accessorius. *Microscopically* the vessels were found to be hypertrophied, and surrounded partly with numerous cells and partly with extravasations of blood. The nervous substance was in a state of inflammational disintegration, and full of pus-cells, corpuscles of Gluge, parenchymatous centres of myelitis (swollen axis-cylinders, etc.)—in fact, a form of *inflammational disintegration with capillary hemorrhage.*

CASE II.—Woman, fifty-two years of age. In the right half of medulla a small centre of softening, situated between the restiform body, the olivary body, and the roots of the hypoglossus, shaped like an olive, without any boundary above or below. Consequently it affected chiefly the roots and nuclei of the vagus, glossopharyngeus and accessorius; the substance of the restiform body, too, was very much involved. *Microscopic examination* showed considerable cellular infiltration, with softening and swelling of the nervous tissues. Cells were large and round, and possessed several nuclei, and there were a few corpuscles of Gluge. The adventitia of the vessels was full of cellular elements. In this case a *commencing inflammational* (embolic ?) *softening.*

CASE III.—Man, sixty-two years of age. Centre of softening in the internal fields of the medulla, extending from the floor of the fourth ventricle into the pyramids, and from the centre of the olivary bodies to its anterior extremity, tapering to a point anteriorly and posteriorly. *Under the microscope* the structure could hardly be recognized, the tissue showing a tendency to disintegration; few corpuscles of Gluge; reactive demarcation on the periphery, *i. e.*, parenchymatous myelitis, swollen axis-cylinders and cellular infiltration. *Acute softening* of inflammational character ?

These cases undoubtedly prove the existence of recent small centres of inflammation in the medulla, which in two cases occupied the internal, and in the other the lateral field. Further observations must of course be awaited, bringing with them more knowledge.

Symptoms.

Taking up these three cases, we can draw up the following *description of the disease:* The *commencement* appears to be always acute and rather sudden, accompanied by headache, giddiness; occasionally, too, by vomiting or violent singultus, difficulty in swallowing and speaking, paræsthesia, and symptoms of weakness in the head and extremities. But there is never any disturbance in the sensorium, loss of consciousness, or apoplectic attack.

After a short time more or less developed and severe *symptoms of bulbar paralysis* make their appearance, which affect the different functions of the bulbus in different degrees, according to the situation of the lesion.

In one case swallowing was impossible, in the other the patient could only swallow with the greatest difficulty. Then, again, the tongue is either paralyzed or almost unaffected, whilst articulation is more or less disturbed. Symptoms of paralysis in the region of the inferior facialis and in the soft palate varied in intensity, and were sometimes bilateral, sometimes more unilateral. Usually, at quite an early date, there were very marked symptoms of disturbance of respiration (quick, irregular and halting breathing, dyspnœa, feeling of oppression, cyanosis) and of circulation (rapid, small and irregular pulse, with intermissions, etc.).

Besides this, there were frequent vomiting, troublesome singultus, fits of coughing, violent headaches, small differences in the pupils, etc.

The symptoms are rendered complete by more or less considerable *affection of the extremities*, which can, however, vary greatly according to the position and extent of the lesion; this affection consists in partial paralysis of some or all four extremities, sometimes in hemiplegia (when the process approaches or involves the pyramids). When the pyramids are spared we have no symptoms at all of paralysis, or very trifling ones. In one case signs of loss of co-ordination were observed (destruction of the fibræ arcuatæ ?).

Sensibility was objectively scarcely affected; subjectively complaints were made of pains and formications in the limbs (funiculi restiformes involved ?).

The bladder and rectum may become paralyzed at a later stage. In Cases 2 and 3 there was a slight fever, 38.8° C. (97.9° F.)

Neither epileptic convulsions nor tonic or clonic contractions of the muscles have yet been recorded.

In its further *course* the disease makes rapid progress; the disturbances of circulation and respiration increase; the patient becomes unconscious, and plainly collapses; death results from paralysis of respiration and asphyxia.

The *duration* of the disease is short (four, six, or ten days).

It is not decided whether death is the one invariable *result* of the disease. For the present we should scarcely venture to make the diagnosis until we obtain a post-mortem examination, in order thus to fix the symptoms and render the diagnosis possible in succeeding cases. It will not be possible, before this has been done, to say whether other cases (as is quite possible and even probable) take a more favorable course and result in convalescence. At present nothing certain is known of this.

Diagnosis.

It is certainly not very difficult to recognize an "acute bulbar paralysis," *i. e.*, an acute pathological process in the medulla oblongata. But it is really difficult to say what the process is, and to distinguish it from hemorrhage, embolism, or rapidly developing myelitis from compression.

The following points may perhaps be taken as indicative of primary acute myelitis bulbi: no foregoing or initial symptoms, no loss of consciousness, no convulsions, and moderate fever; rather gradual development of the paralysis and the complete set of symptoms, a matter of at least hours or days; rapid change for the worse.

Of course, nothing decisive can be said till we have collected more cases.

Prognosis.

The same may be said of the prognosis, for it is impossible to draw conclusions sufficient to form a prognosis from three cases which all terminated fatally.

Treatment.

The principles, means and methods which we have treated of in the chapter on the therapeutics of acute spinal myelitis (supra section 9), may be applied to the treatment of bulbar myelitis We would recommend, too, comparison with what has been said of the treatment of paralysis ascendens acuta (supra, section 17.

5. *Chronic Progressive Bulbar Paralysis* (Wachsmuth).—*Progressive Muscular Paralysis of the Tongue, Soft Palate, and Lips* (Duchenne).—*Paralysis Glosso-labio-laryngea* (Trousseau).—*Progressive Atrophic Bulbar Paralysis* (Leyden).—*Progressive Bulbo-nuclear Paralysis* (Kussmaul).—*Primary Atrophy of the Motor Nuclei* (Hallopeau).

Duchenne (de Boulogne), Paralysie musculaire progressive de la langue, du voile du palais et des lèvres. Arch génér. 1860. Vol. II. pp. 283 and 431.—De l'électrisation localisée. 2. édit. 1861. p. 621.—3. édit. 1872. p. 564.—*Duménil*, Gaz. hebdom. 1859. 24 Juin. p. 390.—*Trousseau*, Paralysis glosso-laryngea. Clin. méd.—*Wachsmuth*, Ueber progress. Bulbärparalyse u. Diplegia facialis. Dorpat, 1864.—*B. Schulz*, Beitr. z. d. Motilitätsstörungen der Zunge. Wien. med. Woch. 1864. Nos. 38 and 39.—*Gerhardt*, Capillarektasie im Pons Varoli. Jen. Zeitschr. f Med. and Naturwiss. I. p. 196. 1864.—*De Bonnefoy*, De la paralysie glosso-laryngée. Thèse. Paris, 1866.—*Mignard*, De la paralysie dite glosso-labio-laryn. Thèse. Strasbourg, 1867.—*Huber*, Zur Geschichte der Paralys. glosso-lab.-lar. Deutsches Arch. f. klin. Med. II. p. 520. 1867.—*J. Stein*, Doppelseit. Lähmung des Nerv. fac. u. hypogloss. nebst allgem. prog. Muskelatrophie. Ibid. VI. p. 593. 1869.—*Leyden*, Vorl. Mitt. über progr. Bulbärparalyse. Arch. f. Psych. und Nerv. II. p. 423. 1870.—Ueber progr. Bulbärparalyse. Ibid. II. p. 643. 1870; and III. p. 338. 1872.—*Charcot*, Note sur un cas de paralysie glosso-lar. suivi d'autopsie. Arch. de Physiol. norm. et path. III. p. 247. 1870.—*Duchenne* et *Joffroy*, De l'atrophie aiguë et chron. des cellules nerv. de la moelle épin. et du bulbe rhachid. à propos d'une observ. de paral. glosso-lab.-lar. Ibid. III. p. 499. 1870.—*Wilks*, Guy's Hosp. Rep. XV. 1870. p. 2.—*Hun*, Labio-glosso-lar. Paralysis. Americ. Journ. of Insan. 1871.—*Krishaber*, L'anesthésie de la sensib. réflexe des voies aër. et digest. comme signe précurseur de la paral. l.-gl.-lar. Gaz. hebd. 1872. p. 772.—*A. Eulenburg*, Lehrb. d. funct. Nervenkrankh. 1871. p. 549.—*Cheadle*, Glosso-lab.-lar. Paralysis. St. George's Hosp. Rep. V. p. 123. 1871.—*Benedikt*, Elektrotherapie. 1868. p. 217.—Nervenpath. u. Elekt. 1876. p. 640.—Zur Casuistik d. progr. Lähmung d. Gehirnnerven. Deutsch. Arch. f. klin. Med. XI. p. 210. 1872.—Fall von diffus. Neuritis centralis. Ibid. XIII. p. 94. 1874.—*Lockhart Clarke*, Progress. Muscul. Atrophy, etc. Medico-chir. Trans. Vol. 56. p. 103. 1873.—*Friedreich*, Ueber progr. Muskelatrophie, etc. Berlin, 1873. p. 322.—*E. Hoering*, Ueber paralysis glosso-ph.-lab. Diss. Tübingen, 1870.—*Blumenthal*, Réflex. sur la mal. décrite par M. Duchenne, etc. Gaz. des hôp. 1872. No. 113.—*Bourdon*, Études sur les mal. du bulbe rhach. Bull. de l'Acad. de Méd. 1872. No. 7.—Gaz. hebd. 1872. No. 22. p. 354.—*Déchery*, Quelques formes d'atroph. et de paralysie glosso-laryng. d'origine bulbaire. Thèse. Paris, 1870.—*Kussmaul*, Ueber fortschreit. Bulbärparalyse und ihr Verhältn. z. progr. M.-Atrophie. Volkmann's Samml.

klin. Vortr. No. 54. 1873.—*M. Rosenthal*, loc. cit. 1876.—*Leyden*, Klin. d. Rücken. II. p. 509–525. 1876.—*Hammond*, Diseases of the Nerv. System. 6th ed. p. 502. 1876. *Charcot*, Leç. sur les mal. d. syst. nerv. Tom. II. 1877.—*Hitzig*, Fall von angeborn. Bulbärparal. Berl. klin. Woch. 1874. No. 37, p. 465.—*R. Maier*, Fall von fortschreitender Bulbärparalyse. Virch. Arch. Bd. 61. p. 1. 1874. — *Habershon*, Glosso-laryng. Paralysis. Guy's Hosp. Reports. XX. p. 334. 1875.—*Winge*, Case of Duchenne's Disease. Norsk. Mag. for Lägevid. R. 3. B. 4. (Virchow-Hirsch's Jahresberichte for 1874. II. p. 127).—*Marburg*, Zur Lehre von der progr. Muskelatrophie und progr. Bulbärparal. Diss. Breslau, 1874. *Hallopeau*, Des paralysies bulbaires. Thèse de l'agrégat. Paris, 1875.—*Dowse*, Bulbar paralysis. Brit. Med. Journ. 1876. Nov. 11.—*Foz*, Case of Bulbar Paralysis. Brit. Med. Jour. 1876. Nov. 11.—*Fox*, Case of Bulbar Paralysis. Ibid. Nov. 11.—*R. Kayser*, Zur Lehre von der progr. Bulbärparal. Deutsches Archiv f. klin. Med. XIX. p. 145. 1877.—*Poincaré*, Le système nerveux central. 2. éd. Vol. I. p. 291. 1877.—*Grasset*, Malad. du système nerveux. 1878. p. 433.

Introductory. Historical.

The form of disease which we shall discuss in this paragraph, in spite of its relatively short existence in the records of science, has accumulated quite a large literature, and can boast of an equally long list of different names which it has received.

It is beyond dispute to Duchenne that we owe the first description of this disease, as an individual and defined clinical form. It of course stands to reason that the disease was observed by others before this; but the men (Trousseau, Tuerck, Duménil) who observed it did not treat it as a special form. Duchenne at first gave an accurate clinical description founded on a number of cases, but did not know anything of the nature of the pathological lesion. Although often accompanied by progressive muscular atrophy, he regarded the two as totally different. The one disease consisted in a primary *atrophy without paralysis*, whereas the glosso-labio-laryngeal paralysis was a primary *paralysis without atrophy*.

In the next few years a large number of cases were recorded, some of them with autopsies, which did not, however, reveal the real situation of the lesion, but only allowed the assumption of a progressive atrophy of the nerve-roots as foundation for the symptoms. But in the year 1861[1] Baerwinkel casually pointed out that the seat of the disease should probably be looked for in the central nervous system, especially in the medulla oblongata. Schulz (1864) referred it with the greatest distinctness to the medulla, on both sides of the raphe, but restricted it to the region of the facialis. Wachsmuth in his admirable treatise (1864) declared most distinctly, on theoretical grounds, that at the next autopsy the origin of the disease would most certainly be found in the bulbus medullæ; and in place of Duchenne's laborious title he proposed to call it "progressive bulbar paralysis," a name which has since then been universally adopted. Wachsmuth expected to find a lesion not alone of the gray nuclei on the floor of the fourth ventricle, but also a notable change in the olivary bodies. But later investigations only confirmed the former, and this they have done completely. But it was some years before the confirmation came. At an autopsy which Bonnefoy made (1866), he found sclerosis of the medulla with increase of connective tissue; but the investigation, which can only be carried out properly with the aid of modern methods, was very imperfect.

Decisive results were first obtained in 1869, and published in 1870, by Charcot in France, and by Leyden in Germany, after Charcot and Joffroy had pointed out (1869) in two cases of progressive muscular atrophy the signification of the disappearance of the ganglion-cells of the bulbar nuclei in atrophic paralysis of the tongue and lips. The results obtained by Charcot and Leyden agreed pretty well in all important points. The French authors, however, laid particular stress on the atrophy and disappearance of the ganglion-cells, asserting this to be an essential, and sometimes even the primary and only change in bulbar paralysis. Since this further anatomical investigations have been made (Duchenne and Joffroy, Gombault, R. Maier, and others), which merely confirmed the former views without as yet solving the question of the primary or secondary nature of the process in the ganglion-cells of the gray bulbar nuclei.

The numerous cases which have been recorded, and the comprehensive descriptions of the disease, have since then thrown considerable clearness over the matter. Amongst the latter Kussmaul's excellent treatise (translated by the Sydenham Society, 1877) deserves special mention, giving a most thorough investigation of the pathological physiology of the disease. Kussmaul proposes the name of "progressive bulbo-nuclear paralysis," on the ground of the pathological results.

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[1] Schmidt's Jahrbücher. Bd. 110. p. 296. 1861.

It is beyond doubt that even at the present day a number of non-related diseases are classed under this name and its synonyms. So we shall follow the example of the better authors, and include *only one form with perfectly defined clinical symptoms, and with an equally definite anatomical lesion*, under the name of progressive bulbar (or bulbo-nuclear) paralysis.

Adopting this name, we shall describe a form of disease which very frequently takes a chronic course, and is always characterized by the most striking regularity. The following are its CLINICAL SYMPTOMS: *Progressive paralysis and atrophy of certain muscles which derive their nervous supply primarily from the bulbus, i. e., the tongue, the lips, the soft palate, pharynx, and larynx;* and in consequence of this, *disturbances of articulation, speech, mastication, deglutition, and of the voice;* later on, *spreading of the paralysis to other important bulbar functions* (especially the respiratory function), *and frequent combination with progressive muscular atrophy.* The ANATOMICAL BASIS *consists in degenerative atrophy of the gray nuclei in the floor of the fourth ventricle*, and often other *extensive sclerotic processes in the medulla and spinal cord, and degenerative atrophy of the paralyzed nerves and muscles.*

We shall deal in the following paragraphs exclusively with this form. This excludes all those cases which, though they exhibit certain bulbar paralytic symptoms, do not give the pure, defined, and constant symptoms of progressive bulbar paralysis, and are the result of a different anatomical lesion (compression of the medulla, or neoplasmata in it, etc.).

Pathogenesis and Etiology.

There is as yet nothing known of the exact mode in w
the pathological process which we have just des

place, and the special causes of it have been very meagrely investigated.

Under the head of predisposing causes, *neuropathic diathesis*, or general nervous disposition, deserves mention, though it certainly does not play a prominent part.

The *period of life* is of more importance—progressive bulbar paralysis being a disease of maturity and old age, and seldom occurring before the fortieth year; most frequently between this and the seventieth year.

Out of nine undoubted cases there was only one patient under forty; this was a girl of twenty. Hitzig's case of a child six years old most undoubtedly belongs to another class of disease. The case Kayser records of a twelve year old boy is, to say the least, doubtful, and was not confirmed by the autopsy. Even Wachsmuth's case—a girl seventeen years of age—admits of doubt on account of the presence of unusual symptoms (paralysis of the superior branches of the facialis, loss of faradic irritability of the nerves and muscles, absence of any motor disturbances in the tongue); the first mentioned symptom, however, was present in my own case of the girl of twenty. In any case, the occurrence of the disease in young people must be regarded as a great rarity.

The *male sex* seems more liable than the female to the disease; of the cases on record, by far the greater number are males. The disease occurs in *all ranks* of society, from the highest to the lowest, and in every profession.

The *exciting cause* most frequently adduced is *catching cold;* and some of the cases undoubtedly owe their origin to this, and I can produce three observations of my own as witnesses. (The above-mentioned girl of twenty years of age attributed the disease to a severe cold she caught during menstruation; a midwife exposed herself, on the occasion of a troublesome delivery, to a bad cold, etc.)

The same undoubted influence is exercised by violent and continuous *mental excitement*, bodily and mental overexertion, *e. g.*, in nursing patients, excessive mental activity, or straining of the muscles affected in the disease (continuous playing of wind-instruments, long reading with a loud voice, etc.). To the same category belong *restricted circumstances*—stinted nourishment.

It has not yet been proved whether progressive bulbar paralysis ever originates in a slight *shock of the medulla* from a fall, a case of progressive bulbar paralysis at all, it was one complicated by chance by a centre in the pons; and besides, there was softening in the cerebellum, and changes had taken place in the spinal cord.

It is only the most accurate *microscopic examination* which can give at all satisfactory explanation of the process in the medulla. And even this gives very uncertain and meagre results with fresh specimens, chiefly corpuscles of Gluge, increased connective tissue, and here and there an atrophied or pigmented ganglion-cell, or a thickened vessel—seldom more than this.

The localization and extent of the pathological process and its character cannot be distinctly and certainly made out until the organ has been hardened and treated with the usual coloring reagents; when this has been done, the diseased parts may be recognized, from their bad coloring, by the naked eye, or with a pocket-lens.

The microscopic examination of the different cases exhibited various processes and of varied extent (because some of them were complicated); but much, and probably all essential particulars, were common to them all.

The chief point seems to be *a degenerative atrophy of the ganglion-cells in certain gray nuclei of the medulla.* This was observed in every case which was accurately examined.

This affection of the ganglion-cells may, as it would appear, exist quite unaccompanied by other changes worth mention (Charcot, Joffroy, Hallopeau, and others); but in the majority of cases we observe simultaneously an increased growth of connective tissue, atrophy of different nerve-tracts, conglomerations of fat-granule- and other kinds of cells, slight changes in the vessels—in a word, we may observe in the medulla, in varying proportions, all that is described under the name of gray degeneration, sclerosis, or chronic myelitis (Leyden, R. Maier).

As we do not yet possess records of so very many autopsies from which we can draw absolute conclusions, it is still an open question whether the chronic myelitis is an essential or secondary part of the process, whether the atrophy of the ganglion-cells is only the secondary result of this myelitis or a primary change, and merely accompanied afterwards (necessarily or casually) by myelitic changes, and especially by alterations in the interstitial especially on the feet or nates, or even on the head without injury to the skull. And the same may be said of the influence of *syphilis*, *excessive smoking*, and other causes that have been asserted.

It often happens that allied pathological processes spread from the spinal cord into the medulla, and thus combine with progressive bulbar paralysis; this is undoubtedly the case with amyotrophic lateral sclerosis and progressive muscular atrophy. But in many cases no cause for the disease can be asserted.

Pathological Anatomy.

It is by no means an easy matter to detect the changes in the bulbus *with the naked eye.* It frequently happens that for the naked eye there is nothing there to be seen in the fresh state, not the slightest alteration of form, color, or consistency.

Often we can, however, notice that the medulla is reduced in size, or unsymmetrical from general or partial atrophy; here and there, too, an unusual hardness or an abnormal want of firmness.

We can generally detect a slight gray decolorization in some parts of a section, and a cloudiness and want of definition in the structure. But this is not brought out properly till the specimen has been hardened in chromic acid; though even this seldom gives absolutely certain results.

In almost every case, however, *the atrophy and gray decolorization of several nerve-roots given off from the medulla* is very striking; these are often reduced to thin, gray threads, that are hardly visible, and are, of course, extremely different from the nerves that remain unaffected. This is usually most pronounced in the hypoglossal and facial nerves; while in the roots of the accessorius, vagus, and glossopharyngeus it is usually less intense, and often unnoticeable. Still less frequently does it extend to the roots, especially the motor roots of the abducens and trigeminus; and the roots of the acoustic are never affected, or only very exceptionally.

Gerhardt's case, in which he found a small centre of softening in the pons, is not above criticism, and the microscopic examination was very imperfect. If it was a case of progressive bulbar paralysis at all, it was one complicated by chance by a centre in the pons; and besides, there was softening in the cerebellum, and changes had taken place in the spinal cord.

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The microscopic examination of the different cases exhibited various processes and of varied extent (because some of them were complicated); but much, and probably all essential particulars, were common to them all.

The chief point seems to be *a degenerative atrophy of the ganglion-cells in certain gray nuclei of the medulla.* This was observed in every case which was accurately examined.

This affection of the ganglion-cells may, as it would appear, exist quite unaccompanied by other changes worth mention (Charcot, Joffroy, Hallopeau, and others); but in the majority of cases we observe simultaneously an increased growth of connective tissue, atrophy of different nerve-tracts, conglomerations of fat-granule- and other kinds of cells, slight changes in the vessels—in a word, we may observe in the medulla, in varying proportions, all that is described under the name of gray degeneration, sclerosis, or chronic myelitis (Leyden, R. Maier).

As we do not yet possess records of so very many autopsies from which we can draw absolute conclusions, it is still an open question whether the chronic myelitis is an essential or secondary part of the process, whether the atrophy of the ganglion-cells is only the secondary result of this myelitis or a primary change, and merely accompanied afterwards (necessarily or casually) by myelitic changes, and especially by alterations in the interstitial

tissue. But the atrophy of the ganglion-cells
to be the deciding cause in the pathogenesis o
to account for the form taken by the symptom
can speak on this subject with absolute certain
further investigations, to be conducted with the
and specially directed to the solution of these

The changes which take place in the gangli
progressive atrophy and contraction; they b
yellow or brown pigment (yellow pigmentar
nuclei disappear, and finally the cells themselv
at most, some unrecognizable remains, in the
glittering, pigmented masses. If we now e
nuclei, and compare them with normal specim
struck by the decrease in number and size. In
obtain a satisfactory general idea of the inten
the process in the various nuclear structures.

In the individual cases the *surrounding ti*
contain corpuscles of Gluge in varying quan
connective tissue and of the nuclei, and a num
("Spinnenzellen"); hypertrophy and fatty de
vessels; often numbers of corpora amylace
nerve-fibres themselves were atrophied and dim
axis-cylinders were often strikingly massive an
the nerve-fibres lose their medullary sheath, an
pletely with the axis-cylinders. In a relati
Benedikt found changes of an undoubtedly inf
acter—*i. e.*, congestion, hypertrophy of the wa
and numerous "inflammational nuclei," etc.

As regards the *topography of the changes*,
obtained seem to prove that the *nucleus of*
the starting or central point of the disease; it
and more completely than the others. The *nu*
sorius and vagus are next attacked, while the
extend in all cases to the nucleus of the glosso-
nucleus of the facial is attacked at a very earl
that part of it which is connected with th
branches. The present pathologico-anatomi
have not decided whether the point attacke

facialis proper of the latest writers, situated in the anterior portion of the rhomboid sinus, or whether it is the "inferior" facial nucleus in the inferior portion of the sinus assumed by Clarke. The *nucleus of the abducens* is very seldom diseased; the *acoustic nuclei* appear never to suffer, and likewise the *trigeminal nuclei;* still, the motor nucleus of the trigeminus has been often found in degeneration, but only the merest traces of disease in the sensory nuclei.

The root-fibres, which go to the nuclei, appear to be more or less strongly atrophied and degenerated.

Thus, we have to deal mainly with a circumscript centre of disease, which has a tendency to spread gradually. It attacks primarily the gray nuclei situated in the inferior portion of the floor of the fourth ventricle, and extends from here very stealthily in all directions.

As regards the other structures in the medulla, the *olivary bodies* are seldom affected, though R. Maier found them degenerated, and Leyden found corpuscles of Gluge, in one case, in their centre. The participation of the *formatio reticularis* is doubtful and not easy to decide; Benedikt found it universally affected. Still, the nerve-fibres running through it are probably involved in every case. The *pedunculi cerebelli* are never attacked. On the other hand, the *pyramidal tracts* have been often found diseased, full of corpuscles of Gluge, sclerosed, and degenerated; and this degeneration could be generally traced up into the pons, and regularly down into the lateral and anterior columns of the spinal cord; but it is quite possible that these were all complicated cases.

In fact, combinations with spinal diseases occur very often. These spinal diseases are sometimes degeneration of the ganglion-cells of the anterior cornua, such as has been of late regularly found as the lesion in the typical form of progressive muscular atrophy; sometimes simultaneous degeneration of the lateral columns and the anterior cornua, producing the form Charcot has described as amyotrophic lateral sclerosis.

Changes in the nerves and muscles. The *nerve-roots* coming off from the bulbus are almost always very much degenerated, and they exhibit the same degenerative atrophy as peripheral nerves after section, *i. e.*, fatty degeneration and disappearance of the nerve-fibres, growth and sclerosis of the neurilemma, leaving nothing of the roots but tough cords of connective tissue, filled with fat, and preserving generally only traces of their axis-cylinders. These changes, though less in degree, may be traced through the nerves and into their peripheral terminations in the muscles. Still, in one case Charcot found but very slight changes in the root-fibres of the hypoglossus.

The roots of the hypoglossi are usually the ones which suffer most from the disease; then the faciales, and next a part of the roots of the accessorius, vagus, and glossopharyngeus. In complicated cases we find the same changes taking place in many of the anterior spinal nerve-roots.

In the muscles, too, we find a very pronounced atrophy, which varies greatly in extent and intensity. The muscles look pale, reddish, or yellow, or a reddish gray; they are very much diminished in size, striped with fat, and partly changed to fatty and connective tissue. Under the microscope, we see intensive atrophy of the muscular fibrils, and notice their small number, the increase of nuclei and connective tissue, and in some parts masses of fat. According to the unanimous view of all modern authors, the changes are exactly the same as those which take place in progressive muscular atrophy, and they exhibit the same irregularity and want of uniformity in their distribution. The tongue usually suffers most, though cases have occurred in which the degeneration could not be diagnosed with certainty till brought under the microscope. Atrophy in different degrees, too, has been observed in the lips and palate and in the pharyngeal and laryngeal muscles.

We generally see in addition similar atrophy of the muscles of the back of the neck, especially the trapezii, and in complicated cases there is usually progressive atrophy of the whole muscular system. Even in uncomplicated cases we very frequently find the initial traces of atrophy in the small muscles of the hand, ball of the thumb, interossei, etc.

In *the other organs of the body* no changes characteristic of this disease take place. Extreme wasting and emaciation of the whole body may usually be observed, as in all other states of inanition. Frequently centres of pneumonia from foreign bodies are found in the lungs, or we have phthisis, or there are symptoms of some other fatal disease. But we need not enter upon this.

Pathology of Progressive Bulbar Paralysis.

Symptoms.

General Symptomatology.—Progressive bulbar paralysis is almost unexceptionally developed in a most stealthy manner.

Slight *premonitory symptoms* generally precede, but escape observation for a long time. These consist of a slight *feeling of oppression and pain in the back of the head and neck*, *an unpleasant sensation in speaking*, a little *giddiness*, etc.; occasionally we may succeed in detecting a loss of reflex irritability in the pharynx and larynx at an early stage of the disease (Krishaber).

The commencement is occasionally accompanied by violent symptoms, such as a sudden impediment of speech, with difficulty in the movements of the tongue and lips; or we may have impairment of the act of deglutition. These symptoms often appear to result from a slight apoplectic attack, but it is doubtful whether they are real cases of the disease we are discussing.

But usually the symptoms come very gradually and stealthily, and the patient sees without suspicion his trifling difficulties growing worse, till after a somewhat short and unrestrainable course they bring his life to an end after fearful suffering.

At first the reigning symptom is *a progressive weakness of the lingual, labial, palatal and pharyngeal muscles.* This does not develop according to the same rule in every case, the weakness beginning sooner and making more rapid strides sometimes in this and sometimes in that direction. This produces a distinction between the different cases, but this is unessential.

The *difficulty of speech* is generally striking, proceeding from *weakness of the tongue.* The pronunciation of certain letters and syllables which are chiefly produced by the tongue becomes difficult, hindering the articulation and rendering the speech indistinct.

This is soon followed by difficulty and indistinctness of the labial sounds, arising from *weakness and stiffness of the lips.* The speech thus loses still more of its clearness, and at the same time the patient is deprived, in part or whole, of his power to whistle, blow, compress his lips, kiss, show his teeth, etc. This is inseparably connected with a *change in the expression*, the mouth becoming lengthened, and the naso-labial folds deeper, and the whole expression peculiarly lachrymose.

Mastication is impaired at a very early stage, chiefly by reason of the difficulty in moving the tongue and cheeks; a real paresis of the muscles of mastication is rare, and never one of the early symptoms.

After a varying length of time we observe *a difficulty in swallowing.* This is due in the first instance to *paresis of the soft palate*, which renders the voice nasal, and often allows fluids to escape through the nose during the act of deglutition; the pronunciation of certain labial consonants ("b" and "f"), too, becomes more difficult.

The muscles of the pharynx and those that close the larynx become gradually *weaker.* This is betrayed by the increasing difficulty in swallowing, which is sometimes greater with solids and sometimes with fluids; sometimes, too, the food "goes the wrong way," in other words, gets into the larynx, and causes fits of coughing and suffocation.

Even at this stage the patients exhibit an extremely characteristic appearance—their peculiar expression, the contrast between the upper movable and lower paretic portion of the face, the faltering, incomprehensible, nasal speech, and the difficulty in swallowing, all betray the disease at the first glance to a practised eye. The case is completed by atrophy, trembling movements and fibrillar twitchings of the lips; by the grooved appearance and atrophy in the tongue, which can hardly be put out, or lies helpless in the bottom of the mouth, in constant vibration with fibrillar twitchings; by the excessive secretion of a thick, mucous saliva, which the patient cannot swallow, and which he must consequently wipe from his lips constantly with a handkerchief.

But the organs of sense and the intelligence generally remain quite unaffected; the most that has been observed being a slightly lachrymose and easily excitable temper, with an inclination to causeless laughter. Sensibility remains quite undisturbed; even the taste on the tongue is perfectly normal. Reflex action is only diminished in the pharynx and larynx.

Thus the disease follows its slow and generally unremitting onward course. There seldom comes a remission of any length, and still more rarely any improvement. We can clearly see from week to week, or at least from month to month, a change for the worse. The speech becomes more and more incomprehensible, swallowing more difficult, the secretion of saliva greater and more annoying, the patient becomes weaker, while his mental functions and intelligence remain unclouded, giving him a mournfully clear idea of what threatens him, and thus adding another pain to his disease.

As the disease proceeds, we get further symptoms, resulting from the extension of the process in the medulla. Such are *weakness and loss of voice*, caused by paresis and paralysis of the vocal cords and the muscles of the larynx; and then *irregular action of the heart*, retardation or great rapidity and irregularity of the pulse, a feeling of faintness and threatening death, accompanied by great terror; further, we have *respiratory disturbances* in the form of a feeling of oppression, weight on the breast, dyspnœa with actual fits of suffocation, difficulty in coughing, sneezing, and clearing the throat, etc. Finally, we may observe *paralysis of the muscles of mastication;* much more rarely do we see any sensible disturbances in the region of the trigeminus; still less frequently are the *orbital muscles paralyzed*, usually beginning with the abducens.

The contrast is striking between these severe paralytic symptoms and the clearness preserved in the higher functions of the brain and organs of special sense, together with the freedom of the extremities from any but trifling affections. Though the extremities, with the exception of inanition from weakness, rarely suffer from any motor or sensible affections, and more especially any actual paralysis, till the very last stage of the disease, still we may frequently observe the development of a progressive atrophy in the smaller muscles of the hand, causing the interossei to fall in, and diminishing the thenar and hypothenar muscles, etc. But this complication often grows to a complete and general progressive muscular atrophy.

At this stage the patients suffer frightful tortures. Speechless and harassed by salivation and the danger of suffocation, with their intelligence perfectly unclouded, they see the approach of a death from starvation. Every endeavor to still their gnawing hunger is vitally dangerous, and causes unspeakable pain; artificial nourishment seldom gives them long respite. Characteristic of the very last stage comes a state of *intense inanition*, which in one way or another brings about the death of the patient. It is real good fortune for those whose lives end quickly from suffocation, or who are carried off after a short attack of pneumonia caused by a foreign body, or for those who are set free by paralysis of the heart or respiratory organs. Any of these is better than death from starvation. Life frequently terminates with a prolonged agony, often accompanied by a terminal coma, rise of temperature, etc., but generally resulting in a simple extinction of life.

Such is a sketch of the most usual and frequent cases, though it is, of course, subject to many variations, and does not run in exactly the same way with the constant quickness and constant intensity.

From our present experience it is doubtful whether the disease ever resulted in anything but death.

Analysis of the Individual Symptoms.

Initial symptoms. Real *premonitory symptoms* are often wanting, and are generally quite trifling and uncharacteristic. There are occasionally complaints of pains in the head and back of the neck, slight dizziness, and a feeling of compression in the neck and breast, which rarely attains the character of actual dyspnœa. We shall not attempt to decide whether these symptoms are to be regarded as signs of initial hyperæmia, constantly occurring in slight attacks.

Krishaber found in two cases that, many months before any definite paralytic symptoms appeared, the pharynx, larynx, trachea, and œsophagus were strikingly slow to answer reflex irritation, while their tactile sensibility remained unaffected. But does this often occur?

The real symptoms of the commencement of the disease usually consist in a *slight affection of the speech*, the patient becoming tired easily after speaking for some time, and finding difficulty in the pronunciation of certain letters and words. The speech, too, grows objectively less distinct.

This is followed by the first symptoms of a *commencing weakness in the lips and palate;* the expression of the face is altered, the voice becomes nasal, and the patient tires after prolonged chewing and swallowing.

This is the usual order of succession in which the symptoms occur; but occasionally they commence with the lips and palate. Even at this early stage we may often observe an increased secretion of saliva.

The duration of the development of the symptoms varies considerably; sometimes it is rather short after a febrile disease or after a febrile initial stage (?). But it is generally pretty long and gradual, the worst being a few rapid periods. Kussmaul records a rare case, which began with a sudden weakness in one of the above-mentioned muscular regions. Actual apoplexy or acute myelitic processes appear never to give rise to a genuine progressive bulbar paralysis.

It frequently happens that an intercurrent disease induces a very rapid change for the worse.

Affection of Speech.

The affection of speech is usually the earliest symptom, and the one most acutely felt. It consists always and without exception of an incapability (produced by paralysis) to pronounce certain letters (and syllables and words which are formed with these letters). The simple and combined motions of the tongue, lips, etc., necessary to the articulation of the sounds, become difficult or impossible. So it is a mere affection of the articulation. Thus we have to deal with *alalia* (or anarthria). It is never a case of stumbling over syllables (Silbenstolpern), a transmutation and confusion of syllables and words; the words are never forgotten, as in genuine aphasia. It is, in fact, always an *affection of the articulation*, caused by paralysis and atrophy of the muscles necessary to speech.

The course of the affection varies according to the order in which the three important muscular provinces, the tongue, lips, and palate, are paralyzed. According to this varies the order in which the different letters become difficult of pronunciation. Finally, all articulation becomes impossible, and the patients can only produce grunts and groans, and even these very imperfectly when the vocal cords are paralyzed.

Primary *weakness of the tongue* renders the vowel "i" first unpronounceable, as its production requires the greatest raising of the tongue. The tongue, too, is in part or altogether the producer of the consonants "r," and "sch" in particular, and of "s," "l," "k," "g," "t," and finally of "d" and "n."

Weakness of the lips causes the loss chiefly of "o" and "u," and also of "e," whilst "a" can be pronounced almost to the last. When the lips become more affected, "p" and "f" cannot any longer be pronounced, and lastly "b," "m," and "w" are lost.

Paralysis of the palate renders the formation of certain labials still more difficult, because the current of air necessary to form explosives escapes through the nose, and consequently loses nearly all its force. Thus, "b" and "p" are turned into "me" and "ve." If we close the patient's nose, these letters are very much better pronounced (Duchenne). Besides this, the nasal tone is due to the weakness of the palate.

When all these affections come together, the speech grows rapidly more and more indistinct, and becomes at last an impossibility. The words sound quite differently from what they should, and gradually only monosyllabic words can be recognized, till at last even the power of producing these is lost, and the unfortunate creatures can only give forth inarticulate, incomprehensible, grunting sounds.

All these affections may be easily explained either by the lesion in the nerve-nuclei and nerves, or by the atrophy of the muscles, both which causes injure the efficiency of the muscles of speech. Thus we see that the affection of speech depends essentially upon the lesion in the hypoglossal nucleus, and part of the nuclei of the facialis and accessorius. It requires further investigation to determine the extent to which the nuclei of the vagus and glossopharyngeus are implicated.

If the affection spreads further in the accessorius nucleus, the innervation of the larynx and the vocal cords becomes weaker, from which result *hoarseness, weakness of voice, and finally complete aphonia.* In the course of this process the patients are often able to groan loudly, and laugh, sometimes in a peculiarly boisterous manner; but, for ordinary speech, their voice is of no service to them. In such cases the laryngoscope reveals paresis and paralysis of the vocal cords.

Affections of Mastication and Deglutition.

These form the second important group of symptoms in this disease. Here, too, we find variations, according to the succession in the paralyses of the various groups of muscles which take part in deglutition.

Particles of food which get between the lips and teeth cannot be caught up by the tongue, and consequently stick there. This condition is rendered worse when the lips and buccinators are paralyzed simultaneously, in which case the food "falls out" more readily. When the paralysis is more complete, food, especially semi-fluid and fluid, falls completely out of the mouth, which can no longer be properly closed. In the same way, too, saliva flows constantly, and has to be wiped away every moment by the patient.

But when the disease attacks the motor nucleus of the trigeminus, and thus *paralyzes the muscles of mastication*, the final and worst affection of deglutition is produced. This results in exhaustion and increasing loss of power in chewing, until at last the act becomes an impossibility. The patient is likewise unable to close his mouth, or effect the lateral motion of his lower jaw. The utmost he can now do is to take fluid nourishment, and even this he can hardly effect, as deglutition is so difficult.

The first stage of *deglutition* is affected by weakness in the tongue. There is great difficulty in forming the bolus, and pressing it back against the soft palate and into the pharynx. These movements the patient often assists with his hands, shoving the bolus back till it is caught up by the reflex movements of deglutition.

Weakness of the soft palate finds its expression chiefly in the imperfect closure of the choanæ. At first fluids escape through the nose, and, at a later stage, sometimes even solid food. The regurgitation is favored by the action of the inferior constrictors of the pharynx. In this way a considerable portion of nourishment often escapes.

When the pharyngeal muscles become affected the difficulty and danger connected with swallowing a bolus grow greater, for particles of food are caught up in the sulci and bursæ of the pharynx, and collect there and behind the epiglottis too. Sometimes the whole bolus gets fast, and cannot be moved forward or backward, causing great danger of suffocation.

The greatest inconvenience and danger lies in the possibility of *fluids or solid bodies getting into the larynx.* For, by irritation, they produce coughing and attacks of dyspnœa, or, if they get into the bronchi, they give rise to pneumonia. This danger, too, may assume different proportions, according to the muscles which are first and most paralyzed.

If it is chiefly the body of the tongue which is paralyzed, the root cannot be well brought over the fallen epiglottis, and hence the larynx is not completely shut, so that particles of food and fluids easily find their way into the trachea. A solid bolus passes over more easily, as its weight presses down the epiglottis; but fluids enter very easily.

But, for the preservation of the larynx, the purely laryngeal mechanism for closure seems still more important. This is brought about by the accurate juxtaposition of the arytenoid cartilages and the *plicæ* ary-epiglotticæ (v. Bruns). If the muscles which perform this function are weakened or paralyzed (mm. arytænoid. transv., thyreo-ary-epiglott., and thyreo-arytenoid. ext.), the larynx cannot be closed effectively. But, if this function is quite unimpaired, then food cannot easily "go the wrong way," even if the closure by the tongue and epiglottis is very imperfect. In such cases the patients can swallow fluids and semi-solids without much difficulty. But when the laryngeal closure is affected, the danger in swallowing fluids is greater, as they can penetrate through the smallest opening into the larynx. The patients prefer to avoid this risk by taking more solid and drier nourishment, though of course the difficulty of swallowing this is greater.

When the paralysis extends to the œsophagus, deglutition is a complete impossibility, and recourse must be had to the stomach-pump.

The explanation we gave of affections of speech may be applied directly to deglutition. In this case, also, we are unacquainted with the details as to the participation of the nuclei of the accessorius, vagus, and glossopharyngeus.

Affections of Expression.

In addition to the affections of the speech and deglutition, we have in the expression a prominent and characteristic symptom, due to paresis of the inferior branches of the facial. In fact, a practised eye can make the diagnosis from the features alone.

The *orbicularis oris* suffers more than all; while the levatores lab. sup., quadratus, triangularis, and levator menti, the buccinator, etc., are less frequently affected, and never so intensely. These muscles are almost always plainly atrophied, so that the lips look lean, furrowed, thin, and sharp-edged. Sometimes, too, we see fibrillar contractions passing through them.

This paresis deprives the patient of the power to whistle, blow out a light, show his teeth, point his mouth, etc. Besides this, he can no longer keep his mouth closed; but it is always open, with a hanging underlip, and the saliva flows out in a constant stream.

The expression of the face undergoes a peculiar change, the mouth increasing in breadth, and slightly opened, the naso-labial sulci grow more marked, thus producing a sad, lachrymose, and somewhat stupid look, contrasting strongly with the vivacious movement of the features of the upper half, and with the brightness and motion of the eyes.

The lower half of the countenance remains relatively motionless in speaking and laughing, and the laugh degenerates to a peculiar grin.

The superior branches of the facial are usually unaffected, though in very rare cases they have been paralyzed with varying intensity.

The lesion lies, undoubtedly, in an affection of the nucleus facialis. But there are many doubtful points connected with this. In the first place, Why is this nucleus so regularly and so early affected? A glance at Fig. 30 shows us that the nucleus of the facial is rather far removed from the nuclei of the hypoglossus and accessorius, in which the pathological process seems to commence. Lockhart Clarke's assumption of a facial nucleus at an inferior point, about on a level with the hypoglossal (close and external to this nucleus), is undoubtedly more suitable to explain this early participation. The second question—Why is it that the lower branches, and almost always these alone, are the ones that are attacked?—could also be easily answered on the assumption that the process only extends to this "inferior" nucleus, on a level with the hypoglossus, which exclusively controls the inferior branches of the nerve, while the superior nucleus is only rarely affected, and never till a late period. The explanation of this peculiar difference in the behavior of the superior and inferior branches of the facial, which occurs, too, in other cerebral lesions, we must leave to normal and pathological anatomy.

Affection of the Salivary Secretion.

In nearly every case—though there are exceptions—we are struck, at an early period of the disease, by the *constant flow of saliva from the mouth.* It is sometimes very watery, sometimes more consistent with transparent mucus, and must be constantly wiped away. It soaks through the pillow at night, causing the patients great annoyance.

This is primarily and chiefly due to paralysis of the lips and the muscles of deglutition. The saliva is secreted in normal quantity, but can only be swallowed with difficulty, and consequently collects in the mouth. But, the contraction of the lips being imperfect, the saliva flows over them, and out of the mouth. It is easy to prove on one's self that no extra secretion of saliva is necessary, by avoiding deglutition and holding one's mouth open, and inclining one's head slightly forward.

Though the salivation may be thus to a certain extent explained on the ground of paralysis, still it might be asked, is there not at the same time an actual increase of the secretion? And the question in a great number of cases can be answered in the affirmative.

Schulz estimated in one case a six- or eight-fold secretion. Kayser asserts positively that the secretion is increased, but his proofs are more theoretical than actual facts. His reasoning is that the saliva is secreted uninterruptedly during sleep, and that it does not always commence simultaneously with affection of deglutition. In Kayser's case he could increase the secretion by reflex irritation, and stop it temporarily by means of atropine.

Kayser has discussed the question thoroughly, though he has by no means decided it. The large quantity of the secretion excludes the supposition that it is mere "paralytic saliva." In fact, we know so little of this "paralytic" saliva, or of its manner of production, that we can hardly discuss the question. We must consequently assume an irritation of the salivary centres, which, according to Gruetzner's investigations,[1] governs both the fibres of the chorda tympani, and of the sympathetic which go to the submaxillary gland. But he does not attempt to show how this centre of salivation is irritated, when all the other bulbar functions are paralyzed, nor to localize the centre exactly in the medulla.

Affections of the Respiration and Circulation.

The affections of respiration occur chiefly in the terminal stage. They are as yet imperfectly known.

We have already mentioned the various affections of laryngeal innervation, paralysis of the vocal cords, aphonia, and the

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[1] Beitr. zur Physiol. d. Speichelsecretion. Pflueg. Arch. VII. p. 522. 1873.

attacks of coughing and suffocation caused
foreign bodies during eating and drinking.

The participation of the nuclei of the vag
in bulbar paralysis being beyond doubt, wh
close relationship that exists between them a
centre, we can hardly be surprised to find fre
the respiratory mechanism. However, this i
case, and when it occurs, it is at a late sta
When we meet it in an advanced form, we m
end is drawing near.

We often hear complaints of constriction
breast, accompanied by a want of breath w
foundation—affections in fact, connected with
functions of the respiratory centres. But re
nœa, which are occasionally observed, would
think of irritation in the centres.

We may further note various troubles of
weak and powerless *coughing*, partly due to i
tion, and partly to feebleness of expiration a
vocal cords. Then, too, they find difficult
noses, because part of the current escapes t
They seldom *sneeze*, and often find it impossi
of the expiration diminishes with weakness
centres. The loss of motility in the root of
them a similar difficulty in *clearing their thro*
too, complain that they *can no longer smoke*
chiefly on paralysis of the tongue, and of
lower the inferior maxillary, and of the orbic

Finally comes the stage of *weakness an*
respiratory centres, due to degeneration. T
and the oppressive feeling grow worse, while t
nœa accumulate and breathing grows consta
till asphyxia, either slow and resulting from
or acute, occurring in an attack of dyspnœ
the patient. But the slightest disease of the
such as a bronchial catarrh, or pneumonia, of
an earlier end to the patient's life.

Little is known of the *disturbances in the*

There is no reliable record of retardation of the pulse (irritation of the vagus). An abnormally rapid pulse (paralysis of the vagus) has been oftener recorded, rising sometimes, just before death, to 130, 150, and higher, in the minute. The fainting-fits which so frequently occur, and to which the patients occasionally succumb, may fairly be referred to the centres of cardiac innervation.

Disorders of Nutrition.

One of the most constant attendants of progressive bulbar paralysis is undoubtedly *atrophy of the paralyzed muscles.* This is usually most marked in the case of the *tongue.* This member grows smaller, flatter, shrunken, and marked with furrows; it looks limp, and is kept in constant vibrating motion due to fibrillar contractions. We can often, too, plainly detect atrophy of the *lips*, which grow thin like paper, emaciated, and with sharp edges; the skin over them becomes withered, and also exhibits in many cases these fibrillar contractions. Of course, the atrophy must have advanced pretty far before it can be noticed in the lips. Atrophy of the *soft palate* can probably never be recognized with certainty.

Several authors assert that this atrophy is not constant, and Duchenne himself brings the absence of atrophy forward as a distinguishing characteristic of his bulbar paralysis from progressive muscular atrophy. But all modern observers bear witness to the complete falseness of this view. With reference to this question, I have myself compared my notes of nine accurately observed and genuine cases of bulbar paralysis, and found atrophy recorded in every single one; though in one case it was only found in the lips, and not in the tongue. In six of these nine cases, though the bulbar paralysis was far advanced, there was no complication whatsoever with progressive muscular atrophy (an extremely important matter for the question in hand); in two cases there was a commencing muscular atrophy, and in one case it was slightly advanced.

But we must grant without hesitation that the atrophy does not always appear at an early stage, and runs, by no means, a parallel course with the paralysis. On the contrary, the paralysis may have lasted some time before there is any atrophy, or, at least, before it becomes evident. But we should not forget that there may be a very considerable atrophy and degeneration of the tongue and lips before we can detect it by mere inspection. Charcot's case proves quite plainly that the tongue may retain quite a normal appearance, volume, and smoothness, and yet, on microscopic examination, exhibit extensive degeneration of its muscular fibres.

We are thus led to the conclusion that *atrophy is one of the most constant and regular symptoms of bulbar paralysis*, observing at the same time that it does not run parallel with the paralysis. The atrophy, in precisely the same manner as the paralysis, may commence at various points, and progress from these; and if the two do not begin simultaneously and progress in the same direction, this only proves that they are not causally dependent upon one another, but only co-ordinate.

In many cases we meet with local atrophy and fibrillar contractions of the small muscles of the hand. This is the first indication of complication with progressive muscular atrophy, which we shall discuss more fully later on.

It is extremely probable that the atrophy we have described arises from a lesion of the motor nerve-nuclei in the floor of the fourth ventricle, and that its origin is due in great part to degeneration of the large ganglion-cells in these nuclei. In fact, the symptoms of bulbar paralysis themselves supply the best possible argument for the assumption of a nutritive function of at least some of these cells.

One constant and necessary consequence of the disease occurs sooner or later, in the form of *general nutritive disturbances*—a state of extreme emaciation, increasing and finally extreme inanition, all due to insufficient supply of food. It is self-evident that this must be accompanied by great general weakness, even without pronounced paralysis, and that the patients must keep to their beds, being, in fact, scarcely capable of stirring.

There is no *fever* during the whole course of the disease, unless arising from a chance complication.

Among the *rare symptoms* of progressive bulbar paralysis, we may note *affections of the auditory nerve*, consisting of buzzing in the ears and deafness. Whether they really belong to the disease, or are only chance complications, and part of another lesion, which was confounded with bulbar paralysis, we cannot decide. In any case, they take no prominent part.

The same may be said of *paralysis of the orbital* muscles. It is not a usual symptom of progressive bulbar paralysis ; still, it is occasionally recorded. Nor is it at all strange that the motor nuclei of the nerves supplying these muscles, especially the abducens nucleus, should sometimes participate in the disease ; but it is exceptional. I have myself seen cases in which ptosis and paralysis of other orbital muscles were present, but am not quite sure that they were genuine bulbar paralysis.

As has been already observed, paralysis of the *muscles of mastication* is more frequent, resulting from the spread of the disease to the motor nucleus of the trigeminus. *Sensory affections of the trigeminus* are, on the contrary, very rare, occurring either very exceptionally, or perhaps never. The symptoms are a furry feeling and anæsthesia on both sides of the face, and want of sensation in the tongue.

Sensory affections in the trunk and extremities are quite as rare, though there are occasional records of furriness and loss of sensation in the fingers, excentric neuralgia in the extremities, pains in the back and loins, etc.

Paralyses in the trunk and extremities are, however, more frequent. Here and there weakness and paralysis of the cervical muscles, especially the trapezii, and of the shoulder muscles, etc., have been observed. This is usually accompanied by atrophy, and thus forms a gradual transition to progressive muscular atrophy. On the other hand, we meet with paralyses of the lower extremities, with contractures, and increased tendon-reflex, but no atrophy—a complication which we must regard as amyotrophic lateral sclerosis, and which we shall discuss later on.

Little is known of any *vaso-motor* affections. I observed frequent congestions in the head in one case, a very red face, and a feeling of heat in the limbs ; but it was a man who was always subject to these symptoms.

There is no reliable case of diabetes mellitus or albuminuria occurring in bulbar paralysis, though Déchery makes a short reference to one recorded by Voisin.

The symptoms in a pronounced case of bulbar paralysis are so characteristic that they can be taken in at a glance; but still an *accurate objective examination* in such cases is of service in helping to fix definitely, and enlarge our knowledge of, the whole appearance of the disease.

Besides the functional affections which we have described in full, we may be able to detect the presence of *paralysis* in the tongue, lips, soft palate, and sometimes in the vocal cords. The *tongue* lies almost immovable on the floor of the mouth, and can be extended over the teeth only with great difficulty. Its point can no more reach the palate or cheeks, and its root cannot be raised, though there is not much difficulty in drawing it back. In fact, the paralysis is by no means always evenly distributed over the whole tongue, generally affecting first and prominently the internal muscles, the lingual muscles proper.

We described above the paralysis of the *lips*. The *soft palate* loses its tense appearance, while the uvula hangs loosely, though generally quite vertically, and is usually somewhat swollen. During phonation we observe only very slight motion —often less on one side than on the other. The laryngoscope reveals to us the paresis or paralysis of the vocal cords.

The *results of electrical examination* of the paralyzed nerves and muscles is especially interesting, though it is true that most authors up to the last few years have left us very scanty records. As a rule, we find it stated that in progressive bulbar paralysis both the faradic and galvanic irritability are subject to no noteworthy change, or, at the most, a simple decrease, without any qualitative alteration, may be detected in the very last stage.

Thus, Duchenne, M. Meyer, Grasset, and Hallopeau talk of perfectly normal faradic irritability; Rosenthal, Erdmann, and Déchery consider it normal, or only diminished in a few cases; Benedikt found it retained for a long time in the tongue, while it had diminished in the muscles of the face; Wachsmuth found it completely extinguished; Leyden asserts that the diminution of the electric irritability is not noticeable till the very last stages of atrophy, and that the reaction of degeneration was *not* observed;

Kussmaul alone observed in one case, where the faradic irritability of the orbicularis oris was well preserved, that its galvanic and mechanical irritability had increased. Thus, it appears there was a degenerative reaction in this muscle, although it cannot be deduced with certainty from Kussmaul's brief report, and was certainly not recognized as such.

Encouraged by my observations in progressive muscular atrophy (elsewhere reported[1]), and led on by the conviction that the form of degenerative atrophy with which we have to deal in bulbar paralysis would be physiologically expressed by the "reaction of degeneration," I expected certainly to find this reaction in the disease, and my expectation was fully justified by the first pronounced case which came under my observation, and which I examined with great care. I found, in fact, *the most marked reaction of degeneration in direct irritation of the muscles*, both on the chin and in the lips, and even in the tongue. At the same time, however, the electric irritability of their nerves was normal, or but slightly diminished ; so that we have exactly the same form of degenerative reaction which I have described as characteristic of the so-called "middle form" of certain peripheral paralyses (cf. Vol. XI.), and I have no doubt that we shall find the same thing, if we look carefully for it, in all cases where atrophy of the muscles has reached a certain point.

The patient in this case, which I intend to publish elsewhere in full, was a woman, aged sixty-two, who had shown symptoms of progressive bulbar paralysis for a year and a half, complicated in the last three months by a commencing atrophy of the thenar and interosseous muscles. At the time I examined her, the symptoms of bulbar paralysis had reached their most typical appearance. The *faradic examination* revealed no plain diminution of the irritability in the inferior branches of the facialis and muscles of the lips and chin. The faradic irritability of the tongue, both directly and through the nerve, was likewise normal, or perhaps slightly diminished. *Galvanic examination* of the inferior branches of the facialis showed no diminution of irritability, or only a very slight one; nor was the quality altered (cathodal closing contraction was greater than the anodal, the contraction short, like a flash of lightning). Direct galvanic irritation of the muscles, however, revealed the *plainest symptoms of the reaction of degeneration* in the atrophic labial and mental muscles. When the muscles were irritated through the nerves, there was only a slight, though normal, contraction at the cathodal closure with eight cells, and no anodal closure contraction, while there were extensive though sluggish and tonic contractions when the muscles were irritated directly with the same number of cells. The contraction at the anodal closure, too, appeared greater than at the cathodal. Even with six cells these contractions are quite distinct, while through the nerve no effect is produced. Direct irritation of the tongue with a current of four or six cells resulted in evident closure contractions, and most unmistakably greater at the anodal than at the cathodal closure. The sluggish character of the contraction was not distinctly recognized. We found exactly the same condition in the thenar and first interosseous muscles.

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[1] *Vide supra*, Section 16. Compare, too, *Erb*, Ein Fall von Bleilähmung. Arch. f. Psych. u. Nervenkrankh. V. p. 452. 1875.

Considering the great practical and theoretical interest of these results, it would be very desirable to test them further and in a greater number of cases.

Examination of the sensibility usually reveals no disturbance whatsoever. It is quite normal in the particular cases of the face, mouth, and nares. Frequently, however, the superior cervical vertebræ are painful on pressure.

In many cases the *behavior of the reflex actions* is striking. They are often greatly diminished, or even totally extinguished, so that we can touch and irritate (with our fingers or an instrument) the patient's tongue, soft palate, pharynx, and even larynx, without exciting any considerable reflex action, such as the motions of vomiting or coughing. At the same time the patient can feel and localize each touch quite distinctly. Krishaber examined this point very accurately in two cases, and found even the larynx extremely insensitive to the action of caustic. This is, however, not always the case; on the contrary, I have in many of my cases found that the reflex irritability of the palate and pharynx was retained till the later stages of the disease. Still, in others I have seen it extinguished at an early date.

In the case of which I have given the above sketch, I observed very *striking reflex contractions in the muscles of the chin and lips.* When percussed, their whole surface fell into active, short contractions, but the muscles percussed remained perfectly passive. To prove more clearly their reflex nature, I produced these contractions by similar percussion of the nose.

When the cornea was irritated the diseased muscles contracted most actively.

In conclusion, we may call attention to a number of *negative symptoms*, which are also rather characteristic. In the first place we have perfectly normal intelligence, memory, and sleep; unaffected organs of special sense and unaffected movements of the eyes; normal sensibility; absence of motor and sensory affections in the extremities, at least in the first stages of the disease; normal function of the bladder and rectum; unimpaired appetite and good digestion; and finally, no fever.

Course, Duration, and Terminations.

The *course* of progressive bulbar paralysis is always very slow and chronic from beginning to end.

Any alteration of pace in the course is rare, though it may halt for a little, and then go steadily on its way again. There is seldom an improvement of any duration.

Recovery has never been observed, at least when the diagnosis was certain.

As a rule, the course is fatal, and death generally results in a few years (from one to five), either from progressive general inanition and exhaustion, from fits of choking and asphyxia, from an attack of syncope and paralysis of the heart, or, finally, from intercurrent complications, which effect a more rapid termination than the condition of the bulbar paralysis would lead us to expect.

Complications.

By far the most important and frequent complication of progressive bulbar paralysis is the typical form of *progressive muscular atrophy*. This has been very frequently observed, and may occur in two ways. Either an extensive progressive muscular atrophy attacks an already well-developed case of progressive bulbar paralysis, or at least traces of it may be found on the hands and scapular muscles, and fibrillar contractions occur in different parts of the body. Or, on the other hand, we may see a perfect case of progressive muscular atrophy, and after some time atrophic paralysis of the tongue, lips and palate sets in, so that at last the patient dies of typical progressive bulbar paralysis.

It has often been discussed whether the two forms of disease are related, and merely distinguished from one another by their localization and the different directions in which they develop. Duchenne, indeed, who observed the frequency with which they occurred together, asserted that it was a mere combination of two totally distinct diseases. According to him, bulbar paralysis is paralysis without atrophy, and progressive muscular atrophy, on the contrary, atrophy without paralysis.

But the fact alone of their *frequent combination* speaks for their close relationship. A still stronger argument is that *the clinical symptoms exhibited by the affected muscles are perfectly similar;* in both cases we have weakness and atrophy, with predominance sometimes of one and sometimes of the other, attacking the different muscles and sets of muscles in irregular order, and showing a tendency to progressive disease. Besides, the changes *in electrical irritability are identical, and the same may be said of the pathologico-anatomical process in the nerves and muscles*, which is a typical form of degenerative atrophy. And finally, we find *post-mortem exactly the same anatomical changes in the central nervous system*—either a simple pigmentary atrophy of the multipolar ganglion-cells in the gray substance, or a chronic sclerotic myelitis, producing at the same time the destruction of the ganglion-cells.

The great number and unanimity of modern investigations all compel us to regard progressive muscular atrophy as essentially resulting from degeneration and destruction of the large ganglion-cells of the anterior cornua. And in the same manner all carefully made autopsies teach us that the essential lesion in progressive bulbar paralysis is, in all probability, degeneration and destruction of the large ganglion-cells in the motor nuclei of the medulla oblongata.

Now, it cannot be for a moment doubted that the gray motor nuclei of the medulla are prolongations of the anterior gray columns, and that the two are anatomically and physiologically analogous.

We may accordingly assume that *in both diseases the pathological processes are either essentially identical, or at least very closely related*, and that it depends on their localization chiefly whether the result is muscular atrophy or bulbar paralysis, and that consequently these two diseases may combine frequently and in every proportion.

This view has been very beautifully expounded and argued by Kussmaul. Shortly afterwards Hallopeau, on the same grounds as Kussmaul, asserted the relationship of the two diseases, only distinguished by their localization. Perhaps a little too early, he proposes for both diseases the name of "atrophie primitive chronique des noyaux moteurs," and distinguishes a bulbar form (progressive bulbar paralysis), a spinal (progressive muscular atrophy), and a bulbo-spinal form (a combination of the two).

In the preceding paragraphs we have given the views of nearly all modern neurologists, with which we ourselves, too, fully agree on the grounds of the facts we have learned. Even Trousseau in his time declared for the relationship of the two forms, though he did not think they were quite identical. Benedikt can draw no distinction between cases with and those without atrophy. Leyden, Charcot, Poincaré, Grasset and others all agree to the complete unity of the diseases; and Kayser, in a lately published review of the question, takes the same side.

At the same time it should not be forgotten that high authorities do not take this view. In the first rank comes Duchenne, who was the first to set up the distinction we have so often noticed, asserting that in bulbar paralysis there was *only* paralysis. It is, indeed, hardly credible that so distinguished an observer as Duchenne should have overlooked the atrophy, which is at least very frequent. At a later period Duchenne modified his views considerably. He admitted that the anatomical lesion in the two cases was identical (primary atrophy of the large ganglion-cells), and allowed that they were often combined; but still he held fast to his old clinical distinction of paralysis in the one case and atrophy in the other. He tried to get himself out of the dilemma by the hypothetical assumption that there exist two kinds of multipolar ganglia—motor and nutritive, and that in progressive bulbar paralysis exclusively or chiefly the motor ganglia were affected, and in progressive muscular atrophy only the nutritive. Hammond quite agrees with this view.

One could very easily agree to this, and temporarily accept this rather plausible hypothesis, if it only could be proved that progressive bulbar paralysis really exists *without atrophy. But this has never been proved.* Instead of the constant assertion that there is no atrophy, in direct con constantly observed by all modern authors.

The histories of his own cases prove that Duchenne himsel with cases without atrophy. In observations 140 and 141 (Él pp. 622 and 629), Duchenne narrates that the tongue was wri "et en apparence atrophiée," and then goes on naïvely to stat after a few faradic sittings, it again assumed "its normal v have been atrophied before.

It is quite plain that it is not always an easy matter to organ like the tongue, and that further it may be concealed b of connective tissue or deposition of fat. Naturally, too, dist of the tongue, which requires such precision, can be detected a than the atrophy which creeps so slowly after. It is almost s as evidence Charcot's observation, that the tongue may during appearance and proportions, and still, when examined post- have undergone extensive degenerative atrophy. Conseque examination, and *not* the clinical, can alone decide the *absen* all observations restricted to a clinical examination, or which microscope at the autopsy, as well as all which relate to othe erative atrophy of the gray nuclei, are utterly worthless for question.

Nothing can decide but a complete autopsy embracing al *no such an one.* In the only one (which was published by D performed by Joffroy) it is a matter of extreme regret that lips, etc.) could not be examined. The decisive autopsy, com be made.

Accordingly, Déchery's thesis, written probably at least in guidance, and attempting the rescue of "bulbar paralysis *witho* ly be regarded as a success. Déchery brings forward this for to prove its existence, and since he cannot bring himself hypothesis of separate motor and nutritive cells, strives to ge by assuming that in *non*-atrophic bulbar paralysis only a nuclei are affected, and all or a great many in the atroph hypothesis certainly sounds more plausible than this.

But in the last few years the doctrine of close relationshi bulbar paralysis and muscular atrophy has found a stout op He devotes a special chapter, in his work on progressive mus relationship between it and progressive bulbar paralysis, and ing conclusions: Progressive bulbar paralysis is essentially a is characterized clinically by the fact that the muscles retain yet sooner or later atrophy may be superadded. Now, if commences in a case of already developed muscular atrophy, ascending neuritis, derived from the muscles, spreads to the c spinal cord, and is from this propagated to the medulla oblongata, where it becomes established as bulbar paralysis. In cases where progressive muscular atrophy appears as a complication of bulbar paralysis, we must assume that it is primarily a case of *paralysis*, produced by propagation of the bulbar process into the spinal cord, and that then a secondary atrophy commences in the paralyzed muscles, an atrophy, consequently, which has nothing to do with genuine progressive muscular atrophy. Finally, there exists a progressive atrophy of the muscles of the face, palate, and tongue, which is, however, only a part (generally of late occurrence) of a very extensive general progressive muscular atrophy.

It is not in place to discuss these somewhat complicated views of Friedreich's; in fact, it would not be possible without a very thorough exposition of his myopathic theory of progressive muscular atrophy, for which there is not space in this work. We only repeat, that in opposition to Friedreich's view, it must be asserted that the existence of bulbar paralysis *without* degenerative atrophy of the muscles remains to be proved. And further, our own experience does not lead us to believe that the complication of bulbar paralysis with progressive muscular atrophy does not occur by any means unexceptionally through the intermediation of a *paralysis* preceding the atrophy.

With the facts at present before us, we are compelled to adhere to our conviction that paralysis and atrophy are *always* combined in progressive bulbar paralysis, though the relative proportions may vary, *i. e.*, in one case atrophy may occur earlier and be more marked than in another, and *vice versa*. But it does not appear to us allowable to draw a sharp distinction between the two forms of disease on the mere ground of a difference in the degree of atrophy.

The existence of progressive bulbar paralysis *without* atrophy remains to be proved, and when this is done we shall gladly recognize it, and likewise a primary, isolated, progressive atrophy of the lingual, labial, and palatine muscles. For the present we shall restrict the idea of the bulbar paralysis we have described in the foregoing pages to a disease which commences with more or less early and well-marked degenerative atrophy of the muscles, and which is, in this respect, and in its anatomical foundation, closely allied to progressive muscular atrophy.

A second important complication of progressive bulbar paralysis is with *amyotrophic lateral sclerosis*. Bulbar paralysis often occurs in the last stage of this disease, which has been described by Charcot, and causes the death of the patient. In this case, also, it is characterized by the same anatomical lesion —that is to say, degeneration and atrophy of the gray nuclei of the medulla. The disease is further characterized by the symptoms of progressive muscular atrophy in the superior and spastic spinal paralysis (lateral sclerosis) in the inferior extremities. To this class belong a number of cases described as bulbar paralysis or progressive muscular atrophy. It is clearly a complication of a chronic degenerative process in the medulla, with similar extensive chronic processes in both the white and gray matter of the spinal cord.

The anatomical facts and clinical symptoms allow us to conjecture an immediate relationship between the three diseases—progressive bulbar paralysis, progressive muscular atrophy, and amyotrophic lateral sclerosis. But we need more numerous, embracing, and careful observations before we can determine their relations, and either distinguish sharply between them, or unite them more closely.

Theory of the Disease.

Our whole description shows pretty plainly how little we know relatively, and especially how few certain data we have, to found a theory.

One fact is beyond doubt: that in bulbar paralysis we have a progressive paralysis and atrophy of certain muscles and muscular groups, and that this corresponds anatomically to a degenerative atrophy of the muscles and their nerves and nerve-roots, and finally, of their gray nuclei and large ganglion-cells. The functional derangements may be satisfactorily explained by these anatomical changes.

Probably the degenerative atrophy of the ganglion-cells is the primary part of the disease; for, as this condition of the cells is constant, it cannot be the degeneration of the root-fascicles, or any other conducting tract of the medulla, which gives rise to the symptoms.

We are still in darkness as to how this process in the ganglion-cells comes about, and what it actually is—whether it is a primary (interstitial) myelitis with secondary atrophy of the cells, or a primary idiopathic degeneration. This question seems hardly ripe for discussion. Nor can anything more be said of the manner in which the process is carried out.

Furthermore, there is no light on the exact relationship between what is plainly the primary central lesion, and the secondary changes which take place in the nerves and muscles. It seems to us very improbable that a descending neuritis is propagated from the centre in the medulla along the nerves to the muscles, creating in these latter an inflammational atrophy. The centre in the medulla seems too small and limited to meet this supposition, and shows too few signs of actual inflammation. Besides, the changes in the nerves are not proportionate to those in the muscles, and then, too, electric examination proves that the nerves may be quite unimpaired whilst the muscles are far advanced in degeneration. We must consequently regard this degenerative atrophy as neurotic. But there is nothing to explain the relations of the different affected central structures to the origin of the paralysis and the production of nutritive derangements in the muscles and nerves.

We are here again met by the questions we have already discussed and designated as a rewarding object of future investigations, *i. e.*, Are there separate and peculiar motor and nutritive cells? Have the muscles and nerves separate nutritive centres? How must the lesion be located to produce paralysis alone, paralysis with atrophy, or atrophy without paralysis? Do facts allow us to distinguish between a form of bulbar paralysis commencing with paralysis, but leading, without fail, to atrophy (analogous to spinal poliomyelitis ant. chron.), and another form, in which the atrophy is primary, and in which the paralysis is due merely to atrophy (analogous to the typical form of progressive muscular atrophy)?

These and other theoretical questions await an answer, which can only be given by very deep researches. It would appear advisable here to include not only all cases of progressive bulbar paralysis and progressive muscular atrophy, but also those of amyotrophic lateral sclerosis, and poliomyelitis anterior chronica.

Diagnosis.

When bulbar paralysis is fairly developed, the symptoms are so extremely characteristic that it is hardly possible to mistake them. It is unnecessary to repeat the individual characteristics here.

But it is frequently a difficult matter *to recognize the very* *commencement of the disease.* We should here pay attention to any slight difficulty of speech, a certain stiffness of the lips, slight alteration in the expression of the face, to a feeling of pressure and traction in the back of the neck, slight trembling in the tongue when put out, traces of a nasal voice, etc., etc. Any traces of atrophy in the small muscles of the hand are likewise of importance.

It is hardly worth mention that at this stage there is no trouble about distinguishing the disease from a simple angina, or stomatitis simplex.

When the disease is further advanced the great task is to distinguish between "*bulbar*" *paralysis from other causes* and primary progressive bulbar paralysis.

When the bulbar paralytic symptoms (derangement of speech and deglutition, paresis of the lips, paralysis of the tongue, palate, etc.) are only some of many arising from extensive disease in the central nervous system—*e. g.*, disseminated sclerosis, general paralysis of the insane, amyotrophic lateral sclerosis, poliomyelitis anterior chronica ascendens,—then the first matter to be settled is the diagnosis of these diseases from the symptoms that belong to them, in order thus to gain a clear idea of the position occupied by the bulbar symptoms, and of any special process taking place in the medulla.

But it is more important, when the disease is actually localized in the medulla, to make out its nature, whether it is a primary atrophy of the gray nuclei on the floor of the fourth ventricle, or whether it is a different pathological process. The diagnosis must in this case depend upon the most accurate examination possible of the course and development of the disease, and a careful investigation of all, and especially of the unusual symptoms.

The manner in which the disease commences is the best guide to distinguish it from embolism, thrombosis, and hemorrhage in the medulla. These three always commence acutely and suddenly, whereas progressive bulbar paralysis is very gradual. The paralysis is frequently partial, limited to, or predominant on, one side. An apoplectic attack, violent dizziness, or an epileptoid fit often form the onset. Besides, these diseases have no progressive character, but rather a tendency to improve. Nor is there generally any difficulty in recognizing the later results of these processes, such as hemiplegia with contractures, absence of atrophy, stability of the bulbar symptoms.

The differential diagnosis of *tumors* which compress the medulla is of more importance, as their course is usually one of slow development. Here we should note that at first the patient nearly always suffers from violent headaches, dizziness, buzzing in the ears, vomiting, etc. We should also look specially for initial symptoms of irritation or paralysis of the bulbar nerve-roots, especially neuralgia and anæsthesia in the trigeminus, derangements of hearing, spasms and contractions in the face and tongue, paralysis of the abducens and facialis, or of the tongue, with early pronounced degenerative reaction of a complete nature—all this is the more important, the more unilateral the symptoms are. When the compressing body is situated anteriorly, and acts on the bulbar roots of both sides, it is often hardly possible to draw a distinction. Early paralysis of the limbs with contractures is likewise of importance, when the tendon-reflex is increased and there is no atrophy. Then, too, repeated epileptic attacks should be noted, attacks of fainting and temporary amaurosis, and finally all other symptoms which indicate the presence of a neoplasm within the skull (ophthalmoscopic examination, etc.).

It is easy, as a rule, to distinguish bulbar paralysis from a *diplegia facialis* of peripheral origin. The diagnosis may be founded on the complete paralysis of all branches of the facialis, inclusive of the superior ones, as well as on the intense degenerative reaction we generally find, the complete immunity of the tongue, normal deglutition, and the eventual ageusis of the point of the tongue. Nor is there much difficulty with a diplegia facialis of cerebral origin, which may be distinguished by the simultaneous paralysis of the extremities and perfectly unimpaired electric irritability, and also by the manner in which the disease is developed.

We should not, however, forget, when dealing with symptoms even of bilateral paralysis of the bulbar nerves, that all the symptoms of labial, lingual, and pharyngeal paralysis may be caused by *lesions localized more centrally* in the hemispheres, corpus striatum, internal capsule, etc. In such cases we are advised by Joffroy to observe that the paralyses of the tongue, palate, lips, and pharynx are never long complete. It is also characteristic that they arise from two plainly separate unilateral affections, and there is frequently a preponderance on the one side or other ; there is no atrophy ; electrical irritability is quite normal, and there is no reaction of degeneration ; reflex action remains unaltered, or may be increased ; the extremities are likewise paralyzed, without atrophy.

We may mention, finally, that Jolly in a case of extensive cerebral sclerosis[1] saw the gradual development of a bulbar paralysis of most unmistakable nature, though without atrophy of the tongue, and at the autopsy he found no notable pathological change in the medulla, especially no alteration of the nuclei in the floor of the fourth ventricle. This case teaches us that a very similar set of symptoms may be produced by a lesion in the anterior tracts, and that we cannot be too particular in making a differential diagnosis to note every symptom, even those that seem quite insignificant.

Prognosis.

In all the well-observed and accurately recorded cases of genuine bulbar paralysis, there is hardly an instance of recovery, and but seldom a halt in the course, or a transitory improvement. There are, indeed, a number of recorded cases in which considerable improvement took place, and even some cures ; but when we look closely at these records we see that they are not cases of bulbar paralysis at all, or else very doubtful ones. No typical, genuine case has yet been treated with lasting success. I have myself treated numbers of such cases without success ; in fact, the only case which seemed successful, when reviewed more accurately, cannot strictly be included, although it had certainly very strong points of similarity with the typical form.

With our present knowledge we must, accordingly, term the

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[1] Arch. f. Psych. und Nervenkrankh. III. p. 711. 1872.

prognosis absolutely fatal. We can in such cases prophesy the patient's death within a few years. Benedikt's assertion that the prognosis of "progressive paralysis of the cerebral nerves" is more favorable than that of locomotor ataxy, progressive muscular atrophy, etc., applies very well to a series of diseases which Benedikt classifies under this heading, but which have certainly nothing to do with progressive bulbar paralysis.

In some cases a prognosis may be formed from the stage of the disease and its principal symptoms. Intense dysphagia is, of course, an unfavorable sign, and likewise advanced inanition, dyspnœa, and fits of suffocation. In fact, the greater the respiratory derangements, the nearer is the end. Duchenne considered the incapability of lateral motion of the jaw (paralysis of the pterygoids) as a prognostically unfavorable symptom, as in that case the disease is in the proximity of the nuclei of the vagus, and death may soon be expected.

Treatment.

The prognosis we have just given speaks for the bad success of every treatment.

However, it would be quite unjustifiable to renounce every effort to cure the disease. Perhaps time will bring us suitable agents and methods. Perhaps, too, when taken early, the disease is more tractable; and then the transitory successes that are occasionally attained stimulate us to further therapeutic efforts. Certainly the experience we have gained of progressive muscular atrophy is not exactly encouraging.

We have to deal with a chronic, slowly progressive degeneration, which comes on with very slight symptoms of irritation and without any evident inflammational process or growth of tissue, and the proximate and ultimate causes of which are unknown to us. Accordingly, the indication is to improve the abnormal nourishment of the nervous system, and to rouse the regenerative activity—in a word, the indication is "alterative."

In the *earliest stages* we should apply (according to the initial symptoms) gentle derivatives at regular intervals (Kussmaul), bloody and dry cupping, vesicantia, setons, Priessnitz bandages, and shower-baths (a single stream of water should be used very cautiously).

When the disease has developed we should, above all things, endeavor to *regulate the diet* in such a manner as to avoid irritation of the nervous system. The patients should, if possible, give up their work, and avoid all excitement, and, at the same time, take plenty of wholesome nourishment, limiting themselves in alcoholic drinks, and tea and coffee. Care should be taken that they get enough sleep, and they should have plenty of open air, if possible at the seaside or in the mountains.

We may combine with this any course of treatment which tends to a general stimulation of nutrition, and produces a tonic effect upon the nervous system. In this respect I place most reliance upon a cautious *hydropathic treatment*, which must, however, be continued for a long time and with great regularity. But with aged patients this is not very feasible. I think it advisable, too, in suitable cases, to try the effect of warm saline and chalybeate springs, though I should decidedly warn anybody against hot springs, steam-baths, or mud-baths. A very cautious trial of a hot spring (especially in the mountains) might possibly prove of service to old and decrepit people.

But by far the most important indication is *electricity*, which promises more than any other method tried. Unfortunately, however, the results as yet obtained are similar to those of progressive muscular atrophy, not at all encouraging. Benedikt's reported cures are certainly very brilliant, but they plainly relate to other forms of disease. The only two of his cases which really belong to this class remained unaffected by his treatment. However, Benedikt's publication contains many encouraging therapeutical remarks. I myself effected a cure with galvanism in one case which had all the symptoms of bulbar paralysis, but began with severe pains in the head and joints, and, besides, exhibited symptoms of dizziness and buzzing in the ears, so that I was not certain of my diagnosis.

The most effective *method of galvanism* is as follows: galvanize with stabile application transversely through the mastoid processes, and longitudinally through the skull, the so-called galvanism of the cervical sympathetic (anode on the nuchus, and cathode at the angle of the lower jaw); and then induce movements of deglutition (twelve to twenty at each sitting); besides this, apply, according to circumstances, direct galvanic or faradic currents to the tongue, lips, and palate.

I seize this opportunity of correcting an error which has been allowed to run through our literature for several years, the error of supposing that *galvanic production of the movements of deglutition* is due to irritation of the hypoglossus. It is quite incorrect, for it may be easily demonstrated that the current which suffices to produce these movements is by far too weak to irritate the hypoglossus; and that the movements, too, cannot be induced by a single contraction of the tongue (especially by a unilateral contraction); further, that it is more easily effected at other points than the point of irritation of the hypoglossus; and that, finally, actual direct irritation of the hypoglossus, though accompanied by visible contraction of the tongue, produces *no* movement of deglutition. It is evidently a case of reflex action, arising from the sensible nerves of the pharynx and larynx; in fact, physiology teaches us that irritation of the laryngeus superior induces reflex deglutitory movements. If one tries the experiment on himself, there is immediately a sensation produced as if one had a bolus or draught of water in the throat, and this is irresistibly followed by a movement of deglutition.

The best method to produce it is to place the anode on the back of the neck, and then run the cathode rapidly over the lateral surface of the larynx, repeating this after short pauses. A healthy man requires only six or eight cells, but for bulbar paralysis the current must often be much stronger, and the reflex irritability is easily exhausted. To meet this, we must reverse the current, instead of using cathodal closure. This treatment produces some improvement in deglutition at least for a short time.

The electric treatment must be continued for a very considerable time, with from four to seven sittings a week. The duration of a sitting should not exceed four or six minutes. The patients are frequently irritable and sensitive to a galvanic current, so that great caution must be taken in selecting the number of cells.

Medicines taken internally have never produced the very faintest effect. Still, considering their success in other chronic degenerations of the central nervous system, we shall always be inclined to administer nitrate of silver, iodide of potassium, iodide of iron, chloride of gold and sodium, ergotine, belladonna, and preparations of iron and quinine, etc. We wish to warn against the administration of strychnine and phosphorus.

As the derangement of deglutition grows worse, the importance of the *patient's food* increases. At first all food must be finely divided, and very carefully cooked; it must be soft, juicy, and concentrated nutriment. At the same time we should not forget to vary it suitably, and not cause distaste from monotony. Minced meat, beefsteaks of the same, hashes, russoles, etc., beef-tea, milk and eggs, cocoa, soups of revelenta arabica, and prepared leguminosa, etc., form a basis to which may be added all kinds of soft prepared meats, compotes, and vegetables. Wine and beer should only be taken in moderation.

According as the deglutitory paralysis grows worse, special attention must be given to the act of swallowing; help must always be given, and none but easily swallowed victuals be allowed. Afterwards all particles of food must be removed from the pharynx, the root of the tongue and underneath it, and the mouth carefully washed out.

When deglutition becomes impossible, we must feed the patient through a flexible tube; but with many patients this is rendered impossible by violent fits of vomiting and suffocation. In this case nothing is left but the administration of nutritive enemata, such as solution of pancreas, milk and eggs, bouillon, wine, etc. But patients seldom endure this long.

When the only indication is to overcome increasing exhaustion, gastrotomy and introduction of food through the wound might be recommended as a "dernier ressort." We might hope, by this means, to prolong the patient's life for a short time. But if there are threatening signs of respiratory paralysis, the operation would be superfluous.

Fauvel, in one case, proposed and performed tracheotomy to check the fits of suffocation and dyspnœa. If the patient constantly wears a tube, and eventually has the larynx plugged at every meal to meet these choking fits, and to prevent the entrance of food-particles into the trachea, it may do some good.

To check salivation, Kayser recommends atropine (subcutaneous dose, $\frac{1}{16}$ to $\frac{1}{4}$ gr. (0.0004–0.001); internally, $\frac{1}{8}$ to $\frac{3}{16}$ gr. (0.0008–0.0012). We cannot abstain from the use of narcotics, morphine, chloral, etc., when, towards the termination of his disease, the patient is troubled by dyspnœa and sleeplessness.

The usual analeptica and irritantia may be administered in cases of general weakness, syncope, etc. To close the tragic course the physician might well think of euthanasia.

6. *Other Chronic Diseases of the Medulla.*

Samuelson, Königsb. med. Jahrb. I. p. 93. 1859, and Berliner klin. Woch. 1868. No. 27.—*J. Wagner*, Ueber Hirngeschwülste. Diss. Berlin, 1871.—*Cornil* and *Lépine*, Cas de paralys. génér. spin. antér. subaïgue suivi d'autopsie. Gaz. méd. de Paris. 1875. No. 11.—*Hallopeau*, Des paralysies bulbaires. Paris. 1875.

The description of diseases in the foregoing paragraphs by no means exhausts the pathology of the medulla oblongata. There are several other forms of disease, some occurring as part of the phenomena of more extensive diseases of the central nervous system, and some which occur so seldom and have been recorded so badly that they have no practical value, and are, consequently, hardly worth mention. Probably the future will throw much light upon this province, so that this chapter can only be regarded as a temporary refuge for those diseases of the medulla oblongata to which no place has been yet assigned in our nosography.

The literature of this subject is still very small. We give the following extracts:

Sclerotic centres, which are usually met with in the medulla oblongata as one of the many symptoms of *multiple* cerebro-spinal sclerosis. These may occur typically in all shapes and sizes; they are oftenest found on the floor of the fourth ventricle, pressing more or less deeply into the white substance, and affecting the bulbar nuclei in varying degree; but they have also occasionally been observed in the pyramids, olivary and restiform bodies, and in the formatio reticularis. Sometimes, too, they may be met with in the bulbar nerves.

The microscopic appearance of these centres is quite the same as any other, so we may refer to the description on p. 481. The destruction of the nervous substance proper (nerve-fibrils and ganglion-cells) does not appear at all so complete or of the same kind, as in bulbar paralysis, for instance.

The *symptoms* of these bulbar centres play a prominent part in multiple sclerosis, and there seems to be a special connection between the symptoms of bulbar paralysis we so often meet in this disease and these centres in the bulbus. But Jolly's often-cited case proves that this is not necessary, and that the symptoms may be produced by anteriorly situated lesions.

In such cases we often come across a strikingly deceptive likeness to progressive bulbar paralysis; but the paralysis is generally not complete—only a paresis; atrophy of the muscles is rare, and in general the whole development of the symptoms is not so regular as in the typical form of chronic bulbar paralysis. Besides, the diagnosis is readily confirmed by the presence of numerous other symptoms of multiple sclerosis.

Possibly other individual symptoms in multiple sclerosis, such as scanning speech, monotonous voice, paralysis of the facialis, weakness and trembling of the tongue, salivation, auditory derangements, etc., might be referred to the centres in the medulla. We have already discussed the question, and must here again leave it undecided whether this supposition is correct or whether the symptoms are not due to centres situated in an anterior portion of the brain.

It is somewhat more warrantable to connect the derangements of deglutition, attacks of dyspnœa, syncope, cardiac palpitations, and paralysis, etc., which occur in the last stages of multiple sclerosis, with the development of sclerotic centres in the medulla.

Anatomical changes in the medulla occur, too, usually as a part of *dementia paralytica* (chronic myelitis, myelitis with corpuscles of Gluge, gray degeneration); these changes, however, constitute only a very small part of the lesion, which extends over the whole central nervous system in this disease. It is still questionable, and not at all probable, that the frequent and typical derangements of speech are dependent upon the participation of the medulla. However, it is not a matter for discussion here.

When poliomyelitis anterior subacuta assumes an ascending and progressive character, the process in the spinal cord finally attacks the bulbus, and seems to show a special tendency to settle in the gray nuclei in the floor of the fourth ventricle. But we have no decisive pathological facts on this point. If we may draw conclusions from Cornil and Lépine's interesting case, in which unfortunately the exact microscopical examination of the medulla is not recorded, we have a case of chronic inflammational changes with secondary destruction of the ganglion-cells.

When a *progressive myelitis universalis* finally spreads to the medulla, the changes are probably the same, viz.: chronic myelitis varying in extent, but probably not confined to the gray substance, but embracing the white as well; still this is an undecided question.

But the bulbar changes, which take place in the terminal stages of *amyotrophic lateral sclerosis* (and also in the course of *progressive muscular atrophy*) are, as we have already insisted, exactly the same as in progressive bulbar paralysis.

In all these cases the infection of the bulbus is indicated by the occurrence of bulbar paralytic symptoms, which gradually increase till they assume the typical form of progressive bulbar paralysis—derangement of speech and deglutition, and paresis of the lips and palate, increase or decrease of saliva, derangement of respiration, attacks of suffocation, and all the rest. The appearance of the symptoms always betokens the last stage of the disease; the prognosis is generally very serious, and the disease can seldom be checked in its course.

Among the diseases that are well confined to the medulla a *diffuse sclerosis*—chronic myelitis bulbi—has occasionally been observed, sometimes with and sometimes without simultaneous sclerosis of the brain or spinal cord. But details are wanting.

It is hard to say, from J. Wagner's description, whether his remarkable case (tumor-like hypertrophy and toughness of the pons and the whole medulla, more especially on the right side) was a real tumor or hypertrophic sclerosis. It was more probably a genuine neoplasm.

Samuelson observed one case of *circumscript bulbar sclerosis*, consisting of an indurated spot in the left half of the pyramidal substance about the size of a bean. Microscopic examination proved it to be sclerosis (fine fibrils of connective tissue with numerous nuclei and separated nerve-fibrils).

In this case, after repeated sensory and motor derangements had occurred, sometimes in the right and sometimes in the left extremities, he observed a sudden right-sided hemiplegia with apoplectiform symptoms, and accompanied by troubles of deglutition and respiration, and slight derangement of speech. The tongue was straight, and all the anterior cerebral nerves unaffected; retention of urine; feeling of a hoop around the head from the occiput to the forehead; sensibility normal; reflex action increased. Variations in the course of the disease are recorded, but no details given.

7. *Tumors of the Medulla Oblongata.—Neoplasmata in its Substance.*

Abercrombie, Diseases of the Brain. 1845. p. 103.—*Ollivier*, loc. cit. 3. édit. II. p. 514. 1837.—*Levrat-Perroton*, Cas de glycosurie déterminée par une tumeur colloïde renfermée dans le 4me. ventric. Thèse. Paris, 1859. Canst. Jahresber. 1859. IV. p. 254.—*v. Recklinghausen*, Schädelverletzung, Diabetes, Tumor im 4. Ventrikel. Virch. Arch. Bd. 30. p. 364. 1864.—*Mosler*, *Virchow*, Neubildung im 4. Ventr. mit. Diab. insip. Virch. Arch. Bd. 43. p. 225. 1868.—*Virchow*, Die Krankhaften Geschwülste. I. pp. 183, 387, 424; II. pp. 112, 134, 664, etc. 1863–65.—*Ladame*, Symptomatol. u. Diagnostik d. Hirngeschwülste. p. 43. 1865.—*Immermann*, Berl. klin. Wochenschr. 1865. p. 177.—*Joh. Erichsen*, Zur Casuistik der Tumoren des Verläng. Marks. Petersb. med. Zeitschr. 1870. I. p. 105. —*Edwards*, Tumour in the Medull. Oblong. Brit. Med. Journal. 1870. Feb. 5.—*J. Wagner*, Ueber Hirngeschwülste. Diss. Berl. 1871.—*Bourdon*, Études sur les malad. du bulbe rhach. Gaz. hebdom. 1872. No. 22. p. 354. —*Garrod* and *Philpot*, Papillomatous Tumour in the Fourth Ventr. of Brain. Lancet. 1873. Mar. 1.—*Verron*, Étude sur les tumeurs du 4. ventr. Thèse. Paris, 1874.—*Hallopeau*, Des paralysies bulbaires. Paris, 1875.—*Carpani*, Storia clin. d'un caso di tumore d'un peduncolo cerebellare. Lo Speriment. Ottobre. 1876 (Virch.-Hirsch. Jahresber. for 1876. II. p. 111).

Pathological Anatomy—Cases.

Neoplasmata in the medulla oblongata are of very rare occurrence. Ladame only collected nine cases altogether, and most of these were not actually in the substance of the medulla, but only in its vicinity, in the cerebellum, etc., and only secondarily connected with the medulla.

As a matter of fact, it is extremely hard to distinguish between tumors in the medulla itself, and those in the surroundings, which press upon and irritate it. Clinically it is in most cases impossible, as experience teaches us that tumors, for instance, which grow in the cavity of the fourth ventricle, and develop in the direction of the floor, produce exactly the same symptoms as those which grow in the floor itself, for instance, tumors of the ependyma. And as far as the symptoms are concerned, it would seem *a priori* that there can be no considerable difference whether a neoplasm arises *in* the gray or white matter of the medulla and destroys it from within outward, or whether a similar one penetrates from without, dislocating and compressing it, and thus causing myelitis.

Thus, the anatomical and clinical appearances of the two are intimately connected, and we can in great part refer to what has been already said of chronic compression through neoplasmata. It is utterly impossible to draw a sharp distinction, since most of the recorded cases are imperfect, in as far as the tumor has seldom been limited to the medulla, but has generally involved the surrounding corpp. quadr., pons, and cerebellum to some extent.

So we shall merely mention what tumors occur in the medulla, and then give a few short examples, and from these deduce the symptoms. For the future the cases ought to be more accurately observed and recorded.

Perhaps the most frequent form in the medulla is *tubercle;* they may be of any size up to the dimensions of a walnut, especially in the pons portion; there is usually but one, but there may be several small ones. The surrounding tissue is sometimes quite intact, sometimes more or less affected (Ollivier, Erichsen, Abercrombie).

Gliomata and *glio-sarcomata* have been repeatedly found (Virchow), either developed from the ependyma or in the centre of the medulla; their size and position vary.

Myxomata appear never to be seen except on the choroid plexus of the fourth ventricle, and the same may be said of psammomata, which often occur there also, both large and small.

Fibromata have been discovered in many cases (Liouville, Virchow, Carpani), occurring in sizes up to a bean, and growing either in the ependyma or in the medullary substance.

The secondary changes, which the medulla undergoes in such cases, have not been much investigated; in fact, we have but very scanty records of the naked-eye appearances. But we can predicate, almost with certainty, that softening processes often occur, and sometimes extend far beyond the limits of the tumor itself, and that further the lesion is often complicated by apoplexy and thrombosis.

But in general the tumor involves the neighboring parts of the brain, and seldom remains confined to the medulla; thus the pons is usually, the cerebellum frequently, and the corpora quadrigemina occasionally drawn into the process. This of course has a great effect upon the symptoms.

Intense hydrocephalus must be considered one of the most frequent secondary changes caused by tumors in the medulla, and more particularly in the fourth ventricle. It is produced, on the one hand, by compression of the veins, especially of the vena magna Galeni, on the other hand, through the hinderance to the return of the cerebro-spinal fluid into the arachnoid space, caused by the tumor filling up the fourth ventricle and rendering it impassable.

We give a few extracts of cases.

Ollivier, Case 146: *Two tubercles in substance of medulla;* epilepsy for twelve years; an aura of violent singultus before every attack, and feeling of globus in the throat.

Erichsen: Tubercle the size of an almond, covering right half of bulbus along whole length of fourth ventricle, and extending somewhat to the left; right corp. restiforme quite destroyed; headache, dizziness, slight dilation of pupils, nausea; *vomiting and singultus, aphonia, anæsthesia of the right side of the face;* paresis of the right side of palate; tongue unaffected; finally, paralysis of the bladder.

Edwards: Fibro-cellular tumor in the centre of the medulla; convergent strabismus (paralysis of abducens); *weakness in legs; difficulty in swallowing; incomprehensible speech;* later, *vomiting; hiccuping;* complete alalia; dysphagia; abdominal respiration; paralysis of sphincters and extremities.

Virchow (Geschwülste. II. p. 134): *Fibrous hyperplasia of the ependyma* of fourth ventricle, three to four lines thick, resembling a tumor. Wound on head; after nine years, mental depression, loss of memory, *dizziness*, *headache*, diplopia; *frequent* urination (polyuria?); later on, *constant vomiting* and languor; movements of the eyes (?), pupils, and tongue normal; sensibility unaffected; pulse 56; finally, *derangement of speech*, stupidity, etc.

Liouville (quoted in Verron's work, p. 57): *Small fibroma in ependyma* in left side of calamus scriptorius, one centimetre long and a half centimetre broad. The patient, who suffered from heart and kidney disease, had *glycosuria* during life.

Carpani: Elliptical *fibroma in the pedunculus cerebelli*, near the pons, size of a bean; violent *pain on right side of head;* impairment of vision and *hearing on the right side; vomiting; atactic gait;* weakness of extremities on right side; *right-sided paralysis of abducens and facialis.*

J. Wagner: Tumor occupying the right half of pons and medulla, covered with small prominences; this half hypertrophied to twice or four times normal size (very inaccurately described). Violent headache and dizziness; paralysis of the right facialis, abducens, and hypoglossus; nasal voice; paresis of all four extremities; pain in back of neck; hearing impaired on right ride; finally, derangement of speech and asphyxia.

Homolle (quoted in Verron's work, p. 54): *Glioma on the floor of fourth ventricle*, probably commencing in ependyma; very vascular; processes extending into the corpora quadr., optic thal., and left hemisph. of cerebell. *Vomiting; headache;* transitory loss of consciousness; amaurosis; stagnation papilla; *paresis of left facialis; attacks of vertigo;* somnolence; sudden death.

Mosler-Virchow: Large-celled gliosarcoma of ependyma, pedunculated; five centimetres long, occupying the whole ventricle; one to two centimetres in diameter, causing the cerebellum and medulla to swell out. *Vertigo; vomiting; racking headache*, with considerable remissions of some duration; finally, sudden death with *symptoms of suffocation.* Diabetes insipidus in last few years.

Etiology.

As far as we have any knowledge of them, the causes of tumors in the medulla are exactly the same as of any others in the skull, viz.: traumatic influences, syphilis, tuberculosis, etc. So we merely refer to Obernier's article in Volume XII.

Symptoms.

Frequently tumors of the medulla cause no functional derangement, and are only discovered post-mortem; or they may remain for a long time latent, and then suddenly in a few hours or days cause sudden death with symptoms of suffocation.

But, generally speaking, the final catastrophe is preceded by suffering of some duration. We need only give a short sketch of the general symptoms, which display numerous points of similarity to other cerebral tumors.

Among initial symptoms the most prominent are *headache*, occurring generally in separate attacks, reaching in some cases a frightful intensity, and localized in various parts of the head, but generally in the occiput and nuchus; and secondly, *attacks of dizziness*, and frequent, exhausting vomiting. *Tinnitus aurium*, *singultus*, *epileptic fits*, are of rarer occurrence at an early stage. The latter form a very important symptom; they occur either as typical fits or else as mere epileptic vertigo or petit mal.

While these symptoms remain, and often grow worse, a number of others make their appearance, which are calculated to turn our attention to the bulbus as the seat of the lesion. These consist of *paralytic symptoms in the facialis, hypoglossus, and abducens;* and we see *derangements of speech and deglutition*, with *paresis of the palate* and a nasal tone of voice, frequently, too, *hoarseness and aphonia.* Besides these symptoms, there may be more or less extensive *paralysis of the extremities*, either unilateral or bilateral paresis, with or without contractures. It is very characteristic when we see a hemiplegia *alternating with paralysis* of the facialis, abducens, or hypoglossus.

We frequently meet with *derangement of co-ordination*, reminding us of ataxia, but usually partaking more of the character of the uncertain gait belonging to cerebellar diseases—this is especially the case with tumors of the fourth ventricle.

Derangements of sensibility are less marked. They generally consist of pain in the nuchus, back, and extremities, paræsthesia in two similar regions, occasionally anæsthesia in the trunk or extremities. On the other hand, *anæsthesia of one or both sides of the face* is a very important symptom. Reflex action is often increased.

Special local importance attaches to *tinnitus and deafness*, whilst *amblyopia* and *amaurosis* (founded on the *stagnation papilla* recognized with the ophthalmoscope) are almost constant accompaniments of every kind of tumor within the cerebral cavity.

Occasionally *psychical derangements* have been observed, such as loss of intelligence and memory, depression, etc., but this is rather rare.

Special importance attaches to a *constant and lasting vomiting*, violent and lasting singultus, and *frequently occurring epileptoid convulsions*, and attacks of transitory unconsciousness. Glycosuria and polyuria, which have been often observed (especially in cases of tumor of the fourth ventricle), and probably will be still oftener seen when looked for, deserve particular notice.

The *course* of the disease is probably fatal in every case. Still considerable variations, and remissions and cessations of growth for long periods may occur, and thus the disease may extend over a number of years. Usually, however, the course is an uninterrupted one. The headaches and paralytic symptoms grow worse, vomiting becomes incessant, hiccuping is a source of perpetual annoyance, the epileptic attacks occur more frequently, consciousness grows clouded, and then come deliria, and afterwards coma, and usually life terminates with increased respiratory derangement in an attack of suffocation. Death is generally rather sudden.

Very little certainty as regards *the symptoms of special localization* in the medulla can be derived from the few cases on record. In general, we must content ourselves with making a probable diagnosis of a tumor in the medulla from the general symptoms of a tumor in the brain, and the special symptoms of bulbar paralysis. We can seldom estimate its size, or attempt to localize it more accurately. Still this must always be the aim of our diagnostic endeavors, and we possess even at present a few "points de départ."

The most reliable points are, that *tumors in the anterior portion of the medulla*, especially in the pyramids, produce uni- or bilateral paralysis, with contractures and increased tendon-reflex, sometimes also paralysis of the bladder, while sensibility remains unimpaired; and then, besides, we may have the different symptoms of bulbar paralysis.

Tumors on the floor of the fourth ventricle, on the other hand, produce no marked paralysis, and usually no considerable de-

olivary bodies and *reticular formation*, except t
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Diagnosis.

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cases it is simply impossible to draw a dis
acknowledge both parts as affected.

troubles of hearing, glycosuria, amblyopia, and stagnation papilla, epileptic convulsions, etc., seldom or never occur in progressive bulbar paralysis.

It is a far more difficult task, or perhaps an impossibility, to distinguish between *tumors in the substance of the medulla* and those that compress it chronically from without. When unaided by etiological circumstances, we must have recourse alone to symptoms of disease in the nerve-roots (signs of irritation and paralysis in the trigeminus, facialis, hypoglossus, vagus, etc.) in order to form a probable diagnosis, that the tumor is compressing the medulla from without, and is not growing in its substance.

Prognosis.

Like all other cerebral tumors, those of the medulla, with few exceptions (gummata, etc.) are quite hopeless, and are, in fact, worse than the others in so far as an extremely small-sized tumor suffices to produce the most grave consequences in this vital organ.

Though the growth may cease for some time, and striking remissions are recorded, still, when once the diagnosis is certain, we should give a very guarded prognosis. The few, though undoubted cases in which the fully developed symptoms of a cerebral tumor are recorded to have completely disappeared, cannot be allowed much weight.

Treatment.

Treatment is in this case just as hopeless as in all other cases of cerebral tumor, unless it be a syphilitic gumma. To avoid repetition, we again refer the reader to Obernier's article. The physician must, as a rule, confine himself to a symptomatic treatment after he has exhausted all the usual agerts supposed to act upon neoplasmata.

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